The New England
Journal of Medicine

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VOLUME 218 FEBRUARY 10, 1938 NUMBER 6

THE ABSORPTION OF GLUCOSE FROM THE SMALL INTESTINE IN DEFICIENCY DISEASE

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IN A previous communication,[1] a simplified apparatus and technic for the study of intestinal absorption in man was described. The principle of the method, developed by Miller and Abbott,[2] consists in the occlusion of the upper part of the small intestine at a measured distance below the pylorus by a rubber balloon connected with the distal end of one of the two lumina of a rubber tube. The second lumen of the tube leads into a number of holes that open above the balloon. Through these holes 100 cc. of 15 per cent glucose solution is introduced into the intestine, where it remains for half an hour, after which the remaining solution is removed by aspiration and repeated washings. The position of the tip of the tube, and thus the length (50 cm.) of absorbing small intestine, is established by x-ray examination before the glucose is introduced. Adequate precautions prevent downward leakage and regurgitation into the stomach. Complete details of this technic are given elsewhere;[1] unless otherwise indicated, they will be referred to below as standard conditions.

It has been shown[1] that the amount of glucose absorbed from concentrated solutions under standard conditions in normal subjects is constant. Moreover, above a certain concentration (10 per cent) the amount of glucose absorbed is independent of the concentration of the solution introduced. Thus Cori's law[3] has been confirmed for man. The normal absorption values under the standard conditions vary from 7 to 9 gm. of glucose, averaging 7.77 gm. The age, sex and weight of the adult subject do not appear to have any detectable influence. Changes in the diet of relatively short duration do not affect the absorption rate.

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RESULTS

The present observations are based on these facts, and deal with the absorption of glucose in pathologic conditions, with the standard technic. Mainly because the procedure of intestinal intubation requires a certain amount of co-operation on the part of the patient, the number of cases that could be satisfactorily examined was small. Of 13 patients, 3 (Cases 1, 2 and 3), had diseases of the alimentary canal in which diarrhea was an outstanding feature and had caused nutritional deficiency symptoms (anemia, pigmentation of the dorsa of the hands, smooth tongue). The other 10 cases exhibited deficiency disease of various types, without gross intestinal lesions, though in some instances with clinical evidence of intestinal dysfunction.

Glucose Absorption in Disease of the Intestine. One patient (Case 1), a sixty-year-old, white, single expressman, had been drinking heavily for twenty years. Four years before entry occasional vomiting and intermittent diarrhea began. Eighteen months before entry he began to lose appetite and weight. On admission he had a temperature of 101°F. and showed emaciation and a dry scaly skin, pigmentation of the dorsa of the hands, atrophy of the lingual papillae, and a distended abdomen. He continued to have fever, and developed roentgenologic signs of infiltration in both upper lung fields and of fluid in the left pleural cavity. The liver was slightly enlarged. There was gastric anacidity after the injection of histamine. The Takata-Ara reaction was strongly positive and the patient had a moderate hypochromic anemia. Diarrheal stools showed an increased content of undigested starch and fat. Roentgenologic studies of the gastrointestinal tract were negative except for a somewhat dilated ileum and hypermotility of the colon. Ten weeks after

entry the patient died, and an autopsy disclosed tuberculosis of the lungs, intestine and peritoneum with marked involvement of the mesentery and its lymph nodes, and fatty degeneration of the liver.

Another patient (Case 2), an intelligent, sixteen-year-old Jewish boy, had suffered from diarrhea and intermittent bouts of fever for seven years. He had failed to grow normally, being only 4 feet, 7 inches tall and weighing only 59 lb. The tongue was smooth, the fingernails were small, and there was no pubic or axillary hair. The liver and spleen were enlarged. The stools contained much mucus and an excess of soaps and undigested starch. The stomach contained free hydrochloric acid. Roentgenologic studies of the gastrointestinal tract showed narrowing of the distal portion of the ileum and of the ascending and transverse portions of the colon. There was a marked hypochromic anemia. This case was considered one of severe chronic ulcerative colitis.

Table 1. *Absorption of Glucose under Standard Conditions in Intestinal Disease with Diarrhea.*

CASE NO.	DIAGNOSIS	GLUCOSE ABSORBED
		gm.
1	Tuberculous enteritis and peritonitis	2.85
		4.85
		4.10
2	Chronic ulcerative colitis	4.10
		3.90
3	Nontropical sprue	3.34
		4.84

The third patient (Case 3) had moderately severe nontropical sprue with diarrhea.

As determined by seven separate observations on these 3 patients, there was a definite decrease in glucose absorption under standard conditions. As Table 1 shows, the values obtained were 2.85 to 4.85 gm., which is significantly below the normal range of 7 to 9 gm.

Glucose Absorption in Deficiency Disease. Five patients with typical pernicious anemia (Cases 4 to 8) were studied, of whom 3 (Cases 4, 5 and 6) were tested both before and during remission induced or maintained by liver-extract therapy. All 3 patients showed a diminished absorption during the active stages of the disease and a return to normal after effective treatment. One patient (Case 8) was examined only during remission and had a normal absorption, as did another (Case 7) in mild relapse with 3,000,000 red-blood cells per cubic millimeter.

A patient with chronic alcoholism (Case 9), showing both polyneuritis and pellagra, had a diminished absorption which became normal after he had been successfully treated for three weeks by injection of liver extract, a nutritious diet and autolyzed yeast. Two patients (Cases 10 and 11) with "alcoholic" polyneuritis were examined after treatment and had a normal absorption of glucose.

In contrast to the observations on these 8 cases, in which the deficiency may be related to some factor associated with or related to the vitamin-B complex, are the results obtained in 2 patients with relatively pure scurvy (Cases 12 and 13). Both showed a normal absorption of glucose.

The data for the chief observations under standard conditions in all 10 patients are shown in Table 2.

Dilution inside the Intestine. In the normal subjects,[1] the 15 per cent glucose solution that had been introduced into the intestine was diluted while absorption was going on by a stream of fluid moving into the lumen. After half an hour the concentration of the glucose solution was found to vary between 3.24 and 2.11 per cent, with an average of 2.50 per cent, or about half the osmotic concentration of the blood plasma. The calculated osmotic pressure of the glucose, plus that of the chlorides and their cations recovered in the diluted glucose solution, was found to be approximately equal to that of the blood plasma. Apparently the normal intestine adjusts its contents so that the final total concentration inside its lumen is in osmotic equilibrium with the blood plasma.

Table 2. *Absorption of Glucose under Standard Conditions in Deficiency Disease.*

CASE No.	DIAGNOSIS	GLUCOSE ABSORBED BEFORE TREATMENT	GLUCOSE ABSORBED AFTER TREATMENT
		gm.	*gm.*
4	Pernicious anemia	5.87	8.92
5	" "	4.67	7.48
6	" "	3.33	8.43
7	" " (mild relapse)	7.07	—
8	Pernicious anemia (remission)	—	9.13
9	"Alcoholic" polyneuritis and pellagra	5.60	8.25
10	"Alcoholic" polyneuritis and achylia	—	9.12
11	"Alcoholic" polyneuritis and achylia	—	7.59
12	Scurvy	8.02	—
13	"	7.96	—

With one exception, this diluting capacity of the pathologic intestine, as in the normal individual, was undisturbed. Indeed, unusually large amounts of isotonic fluid were recovered from the bowel half an hour after the introduction of the glucose. The exception was a patient (Case 6) in whom the glucose concentration half an hour after the introduction of the 15 per cent glucose solution was 3.60 per cent.

Blood-Sugar Values. In the normal subjects[1] the blood sugar was found to rise while glucose was being absorbed from the upper part of the intestine. A fairly typical curve could be constructed that reached its peak after half an hour or an hour. At the peak of the curve the blood sugar was be-

tween 119 and 118 mg. per 100 cc., averaging 134 mg.

In the 6 patients (Cases 1, 2, 3, 4, 5 and 9) whose blood-sugar curve during the absorption was determined, a low sugar absorption was accompanied by a flat blood-sugar curve. The highest blood-sugar value obtained was 125 mg. per 100 cc. after one hour, the lowest 88 mg. In some cases the blood sugar showed no rise above its fasting level. Figure 1 illustrates the upper and lower limits of the blood-sugar values in these patients compared with the normal limits. The amount of glucose absorbed apparently exerts a definite influence on the blood-sugar curve.

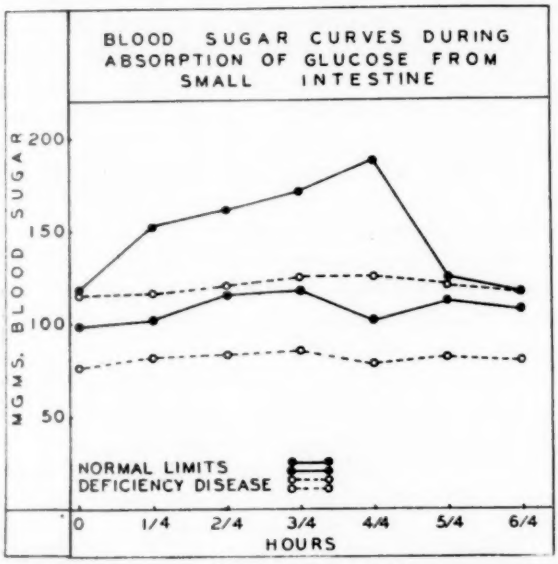

Figure 1. *Upper and lower limits of blood-sugar values following introduction of 100 cc. of 15 per cent glucose solution into a 50-cm. segment of small intestine in normal subjects (solid circles) and in patients with defective absorption (Cases 1, 2, 3, 4, 5 and 9, open circles).*

DISCUSSION

Impaired absorption from the alimentary tract in patients with diarrhea might be supposed to result entirely from too rapid passage of the intestinal contents to allow opportunity for normal absorption. The decreases in absorption demonstrated above cannot, however, have been due to diarrhea only, as the glucose solution necessarily remained above the balloon in constant contact with the same area of intestine. It is therefore clear from the nature of the technic that the absorptive function of the intestinal wall itself was impaired whenever glucose absorption was found to be diminished, not only in the patients with undoubted organic disease of the intestinal tract (Cases 1, 2 and 3), but also in those with pernicious anemia (Cases 4, 5 and 6) and in the patient (Case 9) with "alcoholic" polyneuritis and pellagra. Nevertheless, this feature may be only partially responsible for certain clinical manifestations presented by patients with chronic diarrhea. Thus, loss of body weight and flat blood-sugar-tolerance curves obtained by the usual technic may also be due in part to defective absorption resulting from too rapid passage of material through the intestinal tract.

The occurrence of deranged intestinal function, including poor absorption in certain deficiency diseases with prominent alimentary symptoms, has been suspected for some time.[4] Pierce, Osgood and Polansky,[5] Gál[6] and Westenbrink and Overbeek[7] have shown that rats given a diet deficient in one or more components of the vitamin-B complex absorb glucose from the alimentary tract at a slower rate than do normal controls. Trimble and Maddock[8] found that dogs "in clinically unsatisfactory condition" had a diminished absorption. The work of Gross,[4] McCarrison[9] and Wheeler, Goldberger et al.,[10] which has demonstrated pathologic changes in the intestinal mucous membrane of animals suffering from experimental nutritional deficiency, probably related to the vitamin-B complex, is also pertinent.

In certain human deficiency diseases severe intestinal disturbances occur. In the various forms of sprue the abundant fatty diarrhea and the excess of undigested starch in the stool justify the assumption of an impaired absorption. Less frequently, patients with pellagra, pernicious anemia or other less clearly defined conditions arising directly or indirectly from nutritional deficiency may show similar features.

Poor absorption, especially of glucose, has been assumed by many authors to exist in sprue. Thaysen[11, 12] was among the first to point out that patients with sprue or celiac disease show only a very small rise in blood sugar after the ingestion of 50 or 100 gm. of glucose by mouth. He doubted, however, whether this abnormal sugar-tolerance curve was due to poor absorption, as he also found abnormally flat curves after intravenous administration of glucose. Other observers,[13, 14, 15] however, including ourselves,[16, 17] have found that blood-sugar curves in sprue after intravenous administration of glucose are uniformly normal. Thus the conclusion is logical that the flat curve following the oral administration of glucose is probably caused by a slow absorption from the diseased intestine. In a certain proportion of cases of sprue the oral administration of large amounts of liver extract is relatively or wholly without effect on blood production as compared with the parenteral administration of the same material in usual doses.[18, 19] Mackie and Pound[20] and Mackie, Miller and Rhoads[21] have demonstrated abnormalities of the intestinal pattern by x-ray examination in cases of sprue and of so-called vitamin-B deficiency.

The existence of impaired absorption also suggests itself in those cases of pernicious anemia which require unusually large amounts of liver extract administered by mouth for their recovery or maintenance. Castle and his associates,[22, 23] for example, have presented evidence of this type, and consider that the presence of intestinal stenoses or short circuits may, through interference with absorption, account for the development of macrocytic anemia responding to the parenteral administration of liver extract. The association of the clinical manifestations of pellagra, beriberi and vitamin-A deficiency with disorders of the intestinal tract also suggests malabsorption. Keefer and his associates[24, 25] have noted the development of both hypochromic and macrocytic anemia and signs of vitamin-A-deficiency disease in cases of chronic dysentery. In many of these patients, however, the presence of intestinal lesions was associated with more or less restricted dietary regimes. Heath and Fullerton[26] have shown that the absorption of potassium iodide is sometimes greatly delayed in dietary deficiency conditions. The increased tolerance to galactose in pernicious anemia observed by Donath[27] and by Singer and Wechsler[28] has been ascribed to a slow absorption of this sugar from the alimentary canal.

In spite of the foregoing evidence, however, the existence of a diminished absorptive power of the intestinal wall in human deficiency disease needed proof by a direct test capable of excluding the possible influence of increased intestinal motility. Blood-sugar curves and excretion tests performed in the usual manner are all subject to other metabolic influences than absorption alone. The abnormal results of these tests, therefore, did not necessarily indicate a diminished absorption. However, any influence of increased motility of the intestine or of interfering metabolic factors was excluded by the technic employed in our experiments. They constitute apparently the first *direct* demonstration in man that the passage of material through the wall of the small intestine may be impaired in deficiency states. There is no doubt, therefore, that the flat blood-sugar-tolerance curve in sprue, in anemias from direct or indirect dietary deficiency, and in malnutrition is due, at least in part, to physiologic or pathologic changes in the absorbing mechanism per se. At the same time it should be borne in mind that our experiments deal with glucose only; an impaired absorption of this sugar does not necessarily imply that other substances are also absorbed at a slower rate. Since glucose is a readily diffusible substance, however, it is reasonable to suppose that substances of greater complexity or of larger molecular weight would encounter even greater difficulty. This does not imply what is clearly not the case, that the process of absorption is purely a matter of diffusion. It may at present be regarded as a likely supposition that the absorption is defective in deficiency diseases such as sprue, pellagra and pernicious anemia, and in less well-defined deficiency conditions presenting similar manifestations of the alimentary tract. The explanation of this poor absorption requires further consideration.

Achlorhydria apparently does not significantly affect glucose absorption. A patient (Case 2) with diminished absorption secreted free hydrochloric acid. The cases of pernicious anemia in which the absorption returned to normal after treatment still had gastric achlorhydria. One patient (Case 10) who had a normal absorption was known to have had a histamine-refractory achlorhydria of at least two years' duration. Moreover, it was found in normal subjects[1] that the addition of 0.1 normal hydrochloric acid to the glucose solution actually lowered its absorption rate.

Anemia. The decrease in absorption could not be explained by the level of the hemoglobin or of the red-blood cells. A patient with "alcoholic" polyneuritis (Case 9) had no anemia, yet showed a definitely decreased absorption. A patient with anemia due to deficiency of vitamin C (Case 12) had a normal absorption.

Adaptation. Westenbrink[29] has adduced evidence that the absorptive capacity of the intestinal canal is able to adapt itself to the constituents of the food. When rats were fed on a mixture containing glucose as the main carbohydrate, the absorption rate for glucose increased and that for galactose decreased. When the rats were fed on galactose the reverse took place. When they were fed on a diet rich in fat, the absorption for all carbohydrates diminished markedly. According to Westenbrink, the decreased glucose absorption in vitamin-B deficiency is not specific but is the result of the decreased appetite of the animal and the subsequent lower intake of carbohydrate.

To test Westenbrink's explanation on man, we have tried the effect of changes in diet of normal subjects upon the absorption of glucose. No change could be found when the relative amounts of carbohydrate and protein in the diet were varied. One of the subjects took a ketogenic diet containing only 25 gm. of carbohydrate for ten days. No diminution in the absorption of glucose was found at the end of this period.[1] We therefore find it hard to ascribe the decreased absorption in our patients to a loss of adaptation. It is true that the history of our cases revealed poor appetite and an

unsatisfactory diet, but there was no special restriction in the quantity of carbohydrate taken.

Nonspecific Effect. The poor absorption in our cases might be a nonspecific effect due to the fact that almost all the patients had poor appetites and had been taking only small amounts of food and fluids over a prolonged period before the tests were carried out. The poor absorption, in other words, might be the result of a prolonged undernutrition, by which the intestine had lost its ability to absorb a relatively large quantity of glucose such as was administered for the tests. The diminished absorption might also be considered as a nonspecific phenomenon resulting from the general ill-health of the body. These explanations cannot be entirely refuted. Cori[3] has shown that the absorption rate of glucose in the rat decreases markedly after forty-eight hours of starvation. Also, as mentioned before, a flat glucose curve similar to that seen in sprue is found in cases of general undernutrition.

We[1] have been able to perform absorption tests after complete starvation in 2 patients with epilepsy who were otherwise normal, so far as could be ascertained by clinical examination. One of these patients fasted for forty-eight hours, the other for ninety-six hours; the water intake was not restricted during these periods. At the end of the starvation period both showed a normal glucose absorption. It may be argued that forty-eight hours of starvation for the rat is a relatively more prolonged period than a four-day fast for a human subject, and that the starvation period was too short to allow alterations to occur. We have, however, other indications that the decrease of absorption in our patients must be more specific. Both patients with scurvy who were examined had taken a diet that consisted mainly of bread and of lean, excessively cooked meat, a diet deficient at least in vitamin C but not in caloric content. Both had a normal absorption rate of glucose. The caloric content of the diet had not been particularly low in 2 of the cases of pernicious anemia. It seems unlikely, therefore, that a deficient caloric intake or nonspecifically unbalanced diets can account for the impaired absorption found in our patients.

Vitamin-B Complex. On the other hand, the 5 patients (Cases 4, 5, 6, 7 and 9) with diminished absorption fell into the group of vitamin-B-deficiency disease, if one may so consider pernicious anemia and "alcoholic" polyneuritis. In common with patients having pellagra and sprue, such patients frequently present evidence of derangement of the alimentary tract manifested as glossitis, achlorhydria or diarrhea. The parenteral administration of a fraction of liver soluble in 70 per cent alcohol has been shown in each condition to cause prompt improvement of lingual and intestinal symptoms, and if macrocytic anemia is present, to result in characteristically increased blood production. In many cases similar effects are obtained with hog-stomach preparations or with mixtures of beef muscle or autolyzed yeast with normal human gastric juice.[22, 23] In some instances the administration of beef muscle or autolyzed yeast preparations alone produces such effects.[19] These observations have been made while the patients were being maintained on diets devoid of power to induce remission in pernicious anemia. Such diets have contained no meat, fish or eggs and have consisted largely of refined carbohydrates. According to Castle and his associates,[22, 23] the basis of these effects on blood production and glossitis is the presence in liver-extract preparations of a substance or substances produced in vivo from beef muscle or autolyzed yeast (extrinsic factor) by a constituent of the normal gastric secretion (intrinsic factor). Depending on the natural presence of intrinsic factor in the gastric secretion of the patient, a response to beef muscle or autolyzed yeast alone will be obtained. The administration of hog-stomach preparations supplies the extrinsic as well as the intrinsic factor.

It is proper to assume, at least for the purposes of this discussion, that pernicious anemia, sprue, pellagra and "alcoholic" polyneuritis are deficiency diseases due either to a direct deficiency in the diet,[30] or to a virtual deficiency conditioned by failure of gastric secretion or of intestinal absorption of one or more substances belonging to or closely associated with the vitamin-B complex.[23, 31*] At any rate, it is clear that in each of these conditions under controlled dietary circumstances the parenteral administration of liver extract has been shown to have a promptly beneficial effect on glossitis and on blood formation. In many instances the effect on diarrhea is scarcely less striking.

A therapeutic test consisting of the administration of the vitamin-B complex alone or with gastric juice, all other conditions remaining unchanged, could not be carried out on our patients. When treated in various ways, however, some by injection of liver extract, some by mixtures of gastric juice and various sources of extrinsic factor by mouth, all showed clinical improvement. Shortly an increase in appetite took place, and eventually the patients ate more and better food than they had ingested before treatment. The intake of fluid also increased. It is therefore impossible to conclude exactly what constituent caused the im-

*In this communication the designation "vitamin-B complex" is given to a group of substances, constituents of liver, meat, yeast, and so forth, that are soluble both in water and in alcohol and are necessary for the promotion of growth and for the prevention of changes in the skin, tongue, nervous system and blood of mammals. It includes the antineuritic vitamin, flavin, Györgi's B_6, Castle's extrinsic factor and probably other substances.

provement of absorption during remission in these particular cases. By analogy with other clinical observations[19, 22, 23] under controlled conditions referred to above, however, the cases may reasonably be classed as either direct or conditioned vitamin-B-complex deficiency diseases, and may be presumed to have responded favorably to the administration of such substances either in specific therapeutic preparations or in the food included in the improved diets.

Castle[22] has pointed out with reference to the development of pernicious anemia that in a patient with progressively diminishing power of secretion of the intrinsic factor a point would be reached where the extrinsic factor (vitamin-B complex) would no longer be effective in producing adequate amounts of liver extract. This result would diminish the patient's intake of the extrinsic factor through loss of appetite, and thus a vicious cycle would be established, leading to progress of the disease. We wish to suggest that in addition the vicious cycle would involve progressive decrease of the absorptive capacity of the intestine. Not only glucose but other substances might be absorbed with difficulty. If the diet taken by the patient were already poor in vitamin-B factors, poor absorption would decrease the utilization of even this small amount, or of any products of its interaction with residual amounts of the intrinsic factor. At first the intestine may be able, because of its great length, to compensate to some extent for the diminished absorption per unit of surface. In this stage, liver or stomach preparations given by mouth are still to some extent effective and may induce a remission. If intestinal absorption is further impaired, however, or if some additional circumstance (infection) interferes, therapy by mouth may require extremely large amounts of material in order to be effective. Some cases of pernicious anemia, and especially cases of chronic sprue, are apparently almost totally refractive to oral therapy owing to this absorption barrier. Parenteral administration of liver extract should then be resorted to in order to break the vicious cycle.

SUMMARY

By means of a simplified technic of intestinal intubation which had been previously standardized on normal individuals, the absorption of glucose was studied in 3 patients suffering from organic disease of the intestine associated with diarrhea; namely, 1 case of tuberculous enteritis, 1 of ulcerative colitis and 1 of nontropical sprue. In each case decreased absorption was demonstrated.

Among 10 patients with various dietary deficiency diseases, diminished absorption of glucose was found before treatment in 3 cases of pernicious anemia and in 1 case of "alcoholic" polyneuritis with pellagra. Absorption of glucose in these 4 cases returned to normal after treatment. The absorption of glucose was found to be normal after treatment had been applied in 1 additional case of pernicious anemia and in 2 additional cases of "alcoholic" polyneuritis. However, 2 cases of scurvy before treatment showed a normal absorption of glucose.

Since by the nature of the experimental method the glucose solution was maintained in contact with the same portion of the upper small intestine, the diminished absorption was clearly due to defect of the absorptive capacity of the intestinal wall and not to unusually rapid passage of the intestinal contents. In those cases in which the absorption of glucose was diminished, the blood sugar showed a smaller rise during the absorption test than in control observations on normal subjects. The flat blood-sugar-tolerance curve often found in sprue, and sometimes in pernicious anemia and certain other dietary deficiency states, is therefore attributable, at least in part, to a diminished absorptive power of the intestine.

The decreased absorption of glucose in deficiency disease may be a nonspecific phenomenon, perhaps the result of an insufficient caloric supply over a long period of time. It is more likely, however, to be a specific effect due to a lack of some component or metabolic derivative of the vitamin-B complex.

The author expresses his appreciation to Dr. W. O. Abbott, Dr. G. W. Karr, Dr. C. M. Jones, Dr. H. C. Trimble and Dr. W. B. Castle for advice and encouragement in the course of the work.

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A PLASTIC OPERATION FOR THE CORRECTION OF HYPERTROPHY OF THE BREAST

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BOSTON

HYPERTROPHY of the female breast is not an uncommon affliction. It usually involves both breasts, but occasionally it is unilateral. The increase in size is caused partly by an increase in the amount of fat, but may also be accompanied by a variable degree of actual hypertrophy of glandular tissue. Increased obesity often seems to add disproportionately to the size of the breasts, and dietary measures offer only partial relief.

The degree of pendulousness is sometimes increased by lactation; often the patient has strapped her breasts down with a tight brassière in an effort to make them less conspicuous, and this has caused more or less elongation. These large breasts are a source of considerable discomfort, first because of their heavy weight, and further because of the inconvenience of the various types of support designed to hold them in position. Many patients have complained of tight brassière straps over the shoulders, which sometimes cause pain there, occasionally extending down the arms. The excessive weight often causes faulty posture, the patient assuming a slight stoop in an unconscious effort to conceal the breasts and make them less conspicuous. A certain amount of concealment can be accomplished through dress, but even the most skillful designer may fail to obtain the desired result, so that the patient feels deprived of the privilege of wearing certain types of gowns. Finally there is the problem of mental attitude. Some patients are so sensitive and self-conscious about what they feel to be their deformity that they are definitely handicapped in their social contacts, and deprive themselves of many normal activities in which they would otherwise take an active interest.

The accompanying photographs (Figs. 1-5) demonstrate what can be accomplished by plastic surgery. These 5 cases were selected from a group of 15 to represent the different types which may be encountered. A few facts regarding the cases are as follows:

Case 1. A married woman of 35. She had had one child, who was breast fed. The breasts were long and pendulous, with only a moderate amount of fatty tissue. The postoperative photographs were taken 6 months after operation.

Case 2. A married woman of 34 who had had three children. All were breast fed. She had very heavy breasts, composed not only of hypertrophied gland tissue but also of an excessive amount of fatty tissue. The postoperative photographs were taken 6 months after operation.

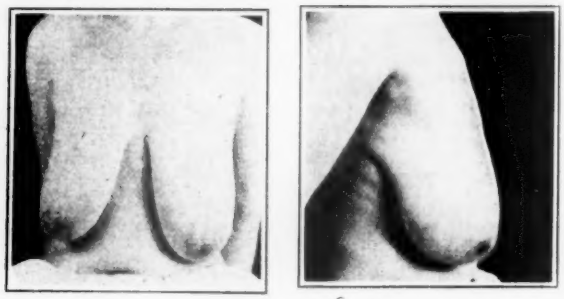
Case 3. A single woman of 25 who had very heavy breasts composed largely of hypertrophied gland tissue. The postoperative photographs were taken 6 months after operation.

Case 4. A single woman of 30. Both breasts were heavy, but there was a very marked glandular hypertrophy on the left side. A plastic operation was done on both breasts, and the postoperative pictures were taken 10 days later. It should be noted that the breasts are quite high; on palpation they were very firm, with areas of induration caused by postoperative inflammatory reaction (some of these areas persist for several months, but eventually disappear).

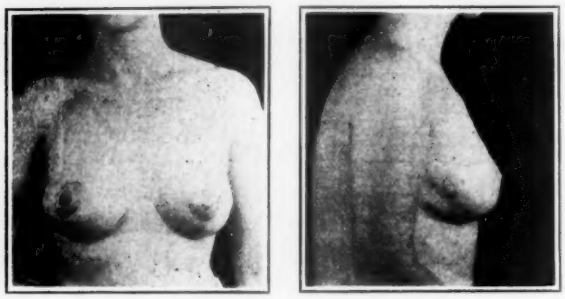
Read before the annual meeting of the New England Surgical Society, October 2, 1937, at Providence, Rhode Island.

From the Surgical Service of the Massachusetts General Hospital, Boston.

*Assistant visiting surgeon, Massachusetts General Hospital.

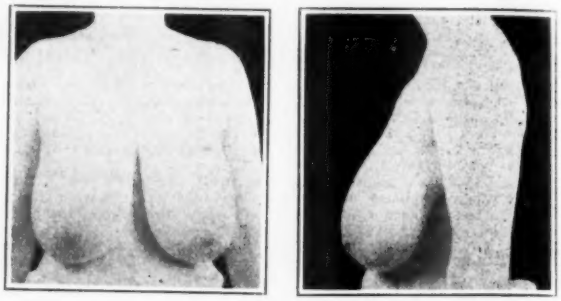


Preoperative.



Postoperative.

Figure 1. *Case 1.*



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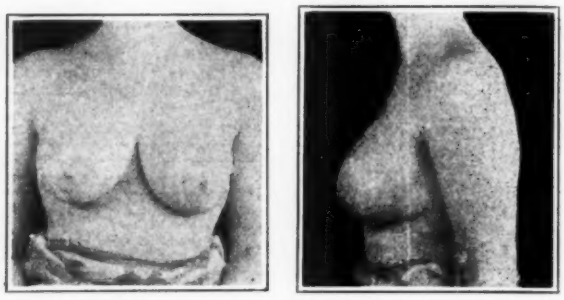
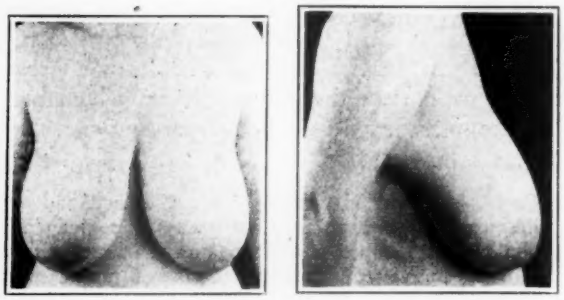


Postoperative.

Figure 3. *Case 3.*



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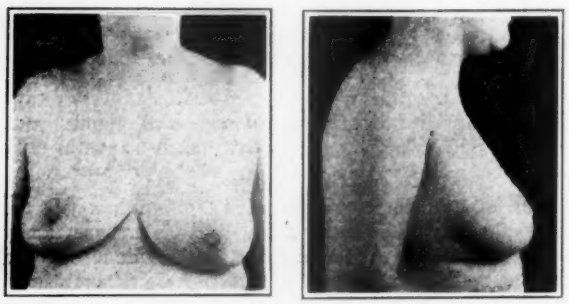
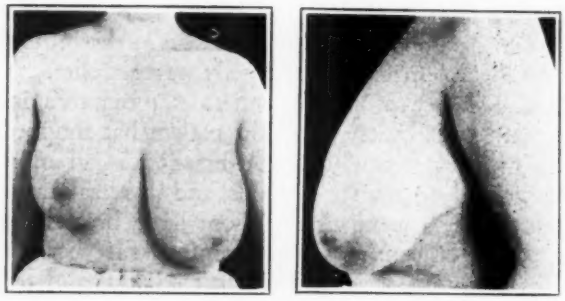


Postoperative.

Figure 2. *Case 2.*



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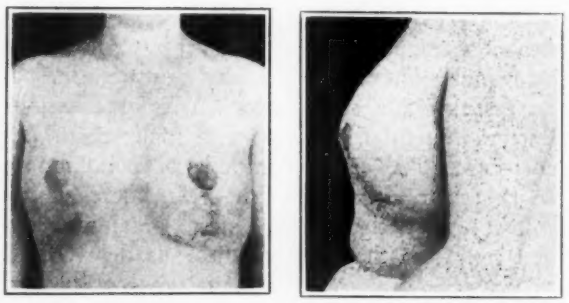
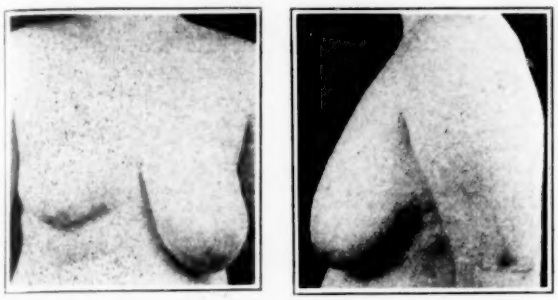


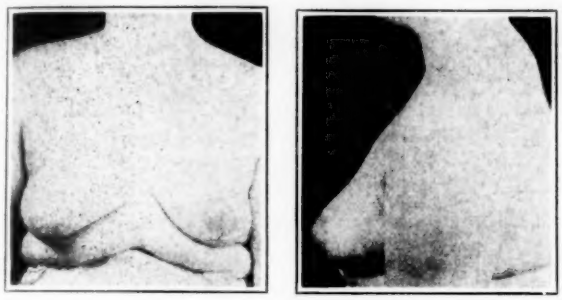
Postoperative.

Figure 4. *Case 4.*

Case 5. A single woman of 26, who had a marked glandular hypertrophy of the left breast. A plastic reconstruction was done. The postoperative photographs were



Preoperative.



Postoperative.

Figure 5. *Case 5.*

taken 5 months later. The normal right breast was of the pointed type, which is difficult to duplicate by operation; however, the side view of the reconstructed breast is satisfactory.

The operation which I have done on these patients is a modification of the Biesenberger[1] procedure, as given in his small but comprehensive book covering plastic and cosmetic operations on the breast. The operation, which he advocated as the most satisfactory, was accomplished by a complete dissection of the skin from the glandular tissue, care being exercised to preserve the nipple with its areola. He then excised the excessive glandular and fatty tissue in order to reduce the breast to the desired size. The breast was reconstructed and sutured to the chest wall, the skin then being fitted around the breast and the excess skin excised at the lower margins. For the details of the operation which I have done I am indebted to Dr. Yolande Huber, of New York City. I have carried it out as follows:

First, a circular incision is made around the nipple at the outer margin of the areola, or if the areola is too large, this incision may be made within it. Another circular incision is made about 0.6 cm. outside the first circular incision. From the lower margin of this outer circle a vertical incision is made down to the submammary fold in the skin below the breast. A horizontal incision from 7 to 10 cm. long is then made along this fold; the skin flaps are carefully dissected from the breast tissue so as to leave the areola and the small ring of skin around it intact, the ring of skin serving temporarily to protect the areola against injury from manipulation. A moderate amount of fatty tissue is left on the under surface of the skin in order that no damage may be done to the circulation of the skin flaps. The entire gland is uncovered so that the pectoral muscle or the chest wall is exposed on all sides of the breast. The breast is then grasped by the apex, and segments of fat and glandular tissue are excised. This excision begins at the areola margin and is carried down to the chest wall; several segments may be removed until the breast is reduced to the desired size. It now appears as a long, thin mass of glandular tissue with the nipple and areola at its apex. In order to reconstruct this into a rounded mass, it is necessary to rotate it upon itself, suturing the folds to each other and suturing the margins to the chest wall. For this purpose fine silk sutures are used. The proper level for the upper margin of the breast is the second rib. The amount of rotation may be as much as 180 degrees. The extra ring of skin around the areola having served its purpose of protecting, is excised, and the skin flaps are carefully fitted around the reconstructed breast. It will be found necessary to excise a considerable amount of skin at the lower borders. No attention is paid to the areola until after the skin has been fitted and sutured. The opening for it is then made at the upper end of the vertical suture line. This opening is circular, and should be at least 2.5 cm. wider than the contracted areola, so that when the latter is sutured to the margins of the opening it will be stretched to its normal size.

It is difficult to describe the operation adequately, but the accompanying group of outline drawings (Fig. 6) in connection with the text will help one to visualize the successive steps.

The postoperative dressing is very important. A liberal amount of gauze dressing is used and over this plenty of sheet wadding; a scultetus swathe is then applied in such a way that it fits firmly and smoothly. Each tail is pinned to prevent loosening, and shoulder straps are attached to prevent slipping. A carefully applied elastic bandage may

be used instead of a swathe. The dressing is left undisturbed for one week.

I have, on several occasions, done this reconstruction operation on both breasts at one sitting, but the procedure is long and tedious, taking from three and one half to four hours. With increased experience in this procedure, I now advise a two-stage operation, with ten days between the first and second stages. I believe this is safer for the patient and probably reduces the chances of infection, although it necessitates the discomfort of two administrations of anesthesia and a ten-day increase in the hospital stay.

As to complications, the extensive elevation of skin predisposes to collections of blood or serum under the flaps, but care in the application of the postoperative dressing reduces this hazard. Only once have I found it necessary to evacuate serum through a corner of the incision, and only once was it necessary to drain an infected hematoma.

This type of operation appears to give the best cosmetic result. In some of the other operations described in the literature, all the tissue is removed from the lower or outer side of the breast. To be sure, this reduces the size and weight, but the end result is a flat type of breast. With the operation described above, the breast is actually reconstructed into a round juvenile type of any desired size, and is then sutured at the correct level to the chest wall.

The question of the possibility of future lactation has been raised. Large, hypertrophied breasts

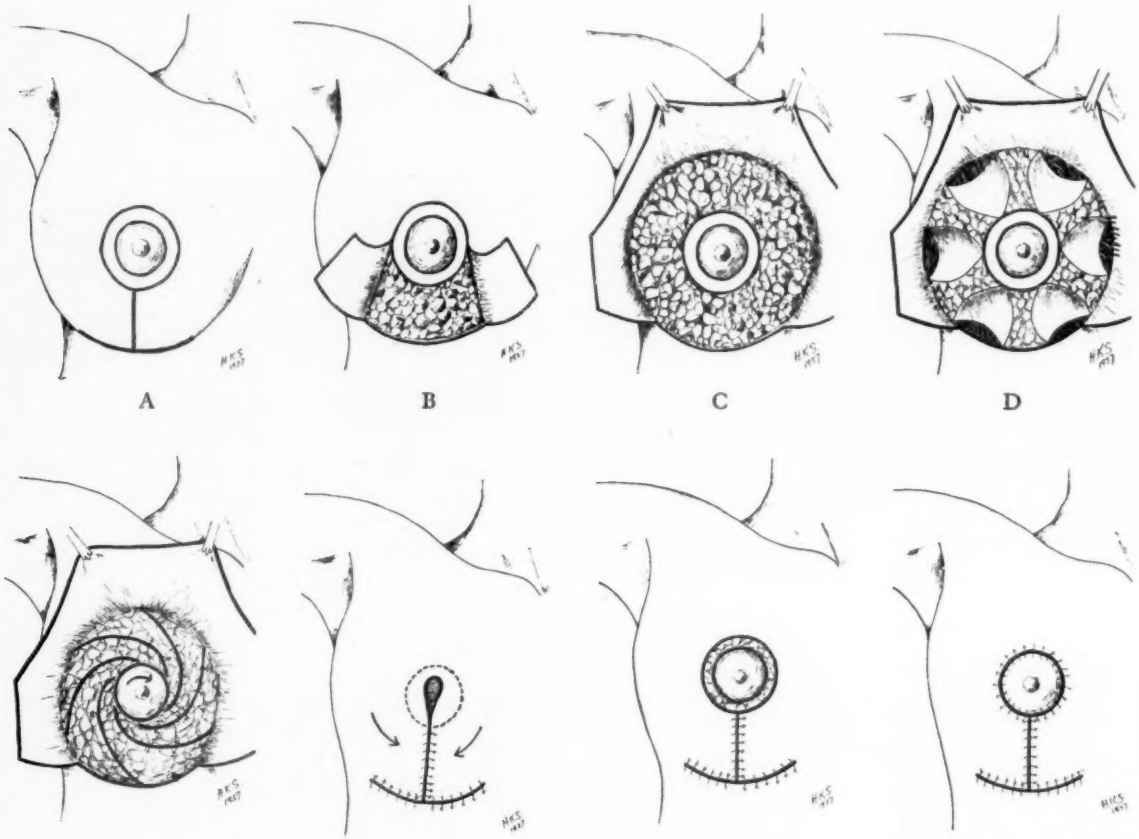


Figure 6.

Drawing A shows the skin incision in heavy ink. Drawing B shows the beginning of the elevation of the skin flaps. Drawing C shows the breast entirely uncovered down to the chest wall on all sides. In Drawing D, the breast is grasped at the nipple and segments are excised from the edge of the areola to the chest wall. In Drawing E, by rotating the gland, a round conical breast is reconstructed; the folds are approximated to each other with fine silk sutures and the margins of the breast are sutured to the chest wall, the upper border being held at the level of the second rib; the extra ring of skin around the areola is then excised. In Drawing F, the skin is fitted around the breast from above downward, the excess being cut away in the midline and below; the areola is temporarily covered. In Drawing G, the opening for the areola is made. Drawing H shows the appearance at the end of the operation.

are notoriously poor as milk-producers. No one of my patients has yet been through a pregnancy, but in all cases both the glandular tissue left behind and the ducts leading to the nipple have been uninjured. I see no reason why these breasts should not function normally, or at least nearly as well as they would have if the operation had not been done.

330 Dartmouth Street.

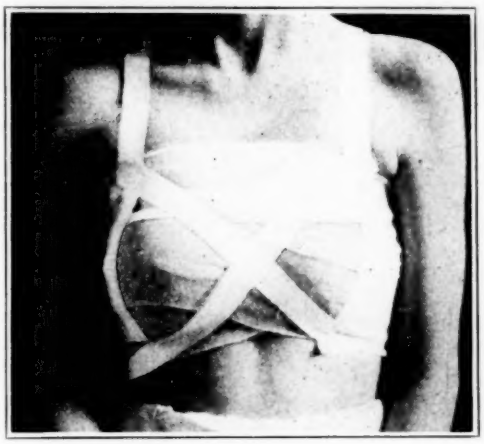
REFERENCE

1. Biesenberger, H.: *Deformitaten und Kosmetische Operationen der Weiblichen Brust.* 205 pp. Wien: Verlag von Wilhelm Maudrich, 1931.

DISCUSSION

DR. GEORGE C. WILKINS, Manchester, New Hampshire: Evidently the women in my locality with large breasts accept the condition with a certain equanimity; at any rate they do not apply to me for relief. The operation described by Dr. Sowles is a very clever one. I know the principles of one or two other procedures in which the skin about the nipple is slid up under a bridge of skin and most of the lower part of the breast is removed. Technically, his method is of course far superior to these, but its success appears to depend entirely upon the most scrupulous attention to details.

DR. CHARLES L. LARKIN, Waterbury, Connecticut: I compliment Dr. Sowles on the splendid results he has obtained. He has emphasized the importance of adequate support of the reconstructed breasts by a properly fitting dressing. I have found that a 3-inch Ace or Bender bandage applied over the breasts and around the thorax in a figure eight



will satisfy all the requirements. It holds the breasts up snugly, allows the patient to breathe easily, and will stay in place for a week or ten days, especially if affixed at the edges with strips of adhesive tape running anteriorly upward over the shoulder and then posteriorly downward.

A NOTE ON RACIAL INCIDENCE IN PORTAL CIRRHOSIS

WYMAN RICHARDSON, M.D.*

BOSTON

FOR some time I have suspected that Italians are peculiarly subject to cirrhosis of the liver. In order to verify this impression, the records of all Massachusetts General Hospital ward patients, during the years 1928 to 1935 inclusive, where the diagnosis of portal cirrhosis appeared, were studied. All cases in which there was a reasonable doubt of severe liver disease, as determined by the usual clinical data, were excluded. A total of 48 cases of portal cirrhosis was gathered. Since there were autopsies in only a few cases, no further attempt was made to differentiate the type of cirrhosis, it being practically impossible to do so without pathological examination. However, all cases suggesting the biliary type of cirrhosis, and all cases with positive blood Hinton or Wassermann tests, were excluded.

The racial incidence among these 48 cases in the total hospital population is indicated in Table 1. It will be noted that 19, or 39.5 per cent, of the 48 cases of portal cirrhosis occurred in native-born Italians, whereas this racial group accounted for only 7.3 per cent of the total hospital population. The incidence of cirrhosis in each of the other groups, including individuals born in the United States of Italian mothers, was lower than that in the total hospital population. Although this is a small series of cases, the number of cases among native-born Italians is sufficiently large to be statistically significant.

Table 1. *Racial Incidence in Cases with Portal Cirrhosis and in the Total Hospital Population.*

RACE	CIRRHOSIS NO. CASES	CIRRHOSIS INCIDENCE	INCIDENCE IN HOSPITAL POPULATION
Italian	20		
Born in Italy	19	39.5%	7.3%
Born in U. S. A. (Italian mothers)	1	2%	5.9%
American	9	19%	32.0%
Foreign (other than Italian)	19	39.5%	54.8%
Canadian	4		
German	3		
Irish	2		
Syrian	2		
English	2		
Chinese	1		
Swedish	1		
Polish	1		
Lithuanian	1		
Armenian	1		
Greek	1		

From the Medical Service of the Massachusetts General Hospital, Boston.
*Associate physician, Massachusetts General Hospital.

It is difficult to account for this apparent racial tendency to develop portal cirrhosis. One naturally associates the disease with the prolonged, though not necessarily excessive, use of alcoholic beverages, and the Italian habit of drinking a peculiar type of red wine may be a possible etiologic factor. The fact that all but one of the patients were born in Italy is interesting, for undoubtedly the drinking habits of the group born in the United States of Italian mothers are quite similar to those of the Italian-born group. Possibly the former have not yet reached the age when cirrhosis is likely to occur.

264 Beacon Street.

AN UNUSUAL CASE OF PAINLESS SWELLING OF THE ANKLE

RUSSELL F. SULLIVAN, M.D.,* HAROLD M. CHILDRESS, M.D.†

BOSTON

PAINLESS swelling of a joint caused by tabes dorsalis or syringomyelia is not infrequent but when a similar joint lesion occurs without demonstrable neurologic or serologic changes, the condition may be classed as a rarity. Interest in this case centers not only upon the pathologic changes in the joint and the primary and secondary factors producing it, but also upon the making of a definite diagnosis. The question arises whether one is justified in calling such joints neuropathic if other evidences of neural lesions cannot be found.

CASE REPORT

The patient, a white male, aged 16, was seen at the Boston City Hospital, November 3, 1936, and complained of painless swelling of the left ankle. He stated that in mid-August, 1935, he had sustained a slight injury to the ankle when he vaulted over a 5-foot fence. It became swollen, but he was able to walk with only moderate discomfort. The day following the injury he attended the outpatient clinic of this hospital, when x-rays were taken (Fig. 1) and the ankle was strapped with adhesive. The x-rays showed a questionable old fracture of the scaphoid with proliferation. He refused further treatment when he was advised to enter the hospital for complete examination. For the next 6 months, he said, he attended school as usual and had no pain in the ankle, but the swelling remained about the same. On February 17, 1936, he was admitted to a local hospital and was subjected to thorough study. Two examiners from the neurologic staff found a diminished sensation to heat, cold and pain over the superficial distribution of peroneal nerves on the left, and to some extent on the right. They decided that this indicated syringomyelia. The patient was discharged 6 weeks later with that diagnosis, and with an arch support for the left foot.

Figure 1. *X-ray film taken shortly after the original injury and showing a lesion in the vicinity of the scaphoid, which was diagnosed as a possible fracture.*

Figure 2. *Appearance of ankle approximately fifteen months after injury.*

This support was discarded in a few weeks. He stated that until his admission, approximately 15 months from the date of injury, he had been performing his usual duties at home and at school without discomfort. The affected ankle, however, had become more enlarged during the last 4 months, and he had been aware upon walking of a vague sensation in that joint.

There was no family history of tuberculosis, cancer or insanity. The patient's health had always been good except for measles, mumps and chickenpox in childhood.

On physical examination the left ankle appeared mark-

From the Bone and Joint Service, Boston City Hospital.
Read before the Boston Orthopedic Club on February 8, 1937.
*Junior visiting surgeon, Bone and Joint Service, Boston City Hospital.
†Former resident, Bone and Joint Service, Boston City Hospital.

edly enlarged (Fig. 2) and the joint cavity was tense with fluid. A definite mass was palpable over the dorsum of the foot at the astragaloscaphoid junction. The circumference of the left ankle was 8.8 cm. greater than that of the right. There was an increase of 6.9 cm. at the midtarsal level of the left foot. All motions of the foot and ankle were limited to 50 per cent of normal. There was moderate tenderness on deep pressure and on forced ankle motion. The ankle joint was not unstable, and no grating was perceptible by either palpation or auscultation. The skin was normal in appearance, and there were no signs of infection or of inflammation.

Examination of the blood showed a hemoglobin of 90 per cent; red-blood cells numbered 4,840,000 and the white

Figure 3. *Later x-ray film showing a definite mass on the dorsum of the foot, resembling an osteochondroma.*

cells 8400. The blood Hinton and Wassermann tests were negative; the urine was negative. The blood calcium was 9.2 mg. per cent; phosphorus 4.8 mg. per cent; phosphatase 0.51 K units. The spinal fluid was clear, with 4 cells per cu. mm.; the pressure and statics were normal; the Wassermann and colloidal gold tests were negative. The Mantoux test was positive at 1:5000. X-rays showed destruction and fragmentation of tarsal scaphoid with proliferation, also proliferation of calcaneal epiphysis (Fig. 3); the diagnosis was neurotrophic arthritis or atypical Koehler's disease.

The joint was tapped, and about 2 oz. of thick, clear fluid was removed. Examination of the joint fluid showed a cell count of 1200: 52 per cent clasmatocytes, 28 per cent monocytes, 16 per cent polymorphonuclears, 4 per cent lymphocytes. Cultures showed no growth. A gonococcal complement-fixation test and a Wassermann test were negative. A guinea-pig injection was negative for tuberculosis.

At biopsy, a thickened synovial membrane was seen, and a large movable cartilaginous mass was found at the junction of the astragalus and the scaphoid. Portions of the synovium, the mobile mass and joint detritus were removed for study. No indication of infection was present. The pathological report was: chronic synovitis; islands of cartilage, some of which are being replaced by bone; some new bone formation with fibrosis of the marrow; no evidence of tumor or tuberculosis; no growth on culture.

In order to determine deep sensibility, three rows of intradermal blebs were made with 1 per cent novocain from the mid-left leg down and over the ankle and foot. One row was anterior, and the others were somewhat mediposterior and lateroposterior. After superficial anesthesia was obtained, a sharp-pointed needle was passed through each bleb into the deep tissue. The charted results showed a definite decrease in deep sensibility, beginning at the level of internal malleolus and extending over the ankle and foot to the level of the base of the metatarsals. Deep insensibility to pain was nearly complete, but the patient felt definite deep-pressure sensation upon needle insertion. The reactions of the right foot and ankle were normal as checked against two controls. These changes in sensitivity may have been caused by chronic distention of the capsule and synovium. As a rule, little deep pain is complained of by patients when having a distended joint aspirated.

In order to substantiate the impression that the condition was caused by persistent trauma to an unprotected insensitive joint, the joint fluid was aspirated and measurements of the ankle were recorded. A Boehler walking cast was applied, and upon its removal 5 weeks later further measurements were made. There was a decrease in the circumference at the level of the scaphoid of 3.8 cm. This evidence was supported when the patient secured permission to return to school for 10 days with the ankle unsupported. He returned at the end of that time with the joint filled with fluid.

The neurological examination was essentially negative. No stigmas of hereditary degenerative diseases of the nervous or skeletal system were found. The joint was neither painful nor abnormally mobile. This may have been a neurotrophic joint, but the absence of neurologic abnormalities elsewhere is against it. The staff concluded after a study of the x-rays and clinical evidence that the condition was one of degenerative fracture of the scaphoid, with subsequent proliferation and displacement of the astragalus.

In a familial neuropathy study the blood serologic tests, spinal tap and neurological examination of the mother were negative. The previous hospital records of a brother were negative for any central-nervous-system complications.

On summing up the information concerning this patient, one feels confident that the condition was neuropathic. Yet confirmation by positive serologic or neurologic changes is desirable, if not essential, for a definite diagnosis. The question remains, Just when is an apparently neuropathic joint sufficiently neuropathic to warrant a final diagnosis as such?

In an effort to avoid an untenable diagnosis we carefully considered a thorough differential. Since within the astragaloscaphoid joint — as noted at biopsy, it was not separated from the ankle joint proper — there was a movable mass, diseases which produce such a lesion was considered. Osteochondritis dissecans and osteochondromatosis were ruled out by x-rays and by examination of the joint and its tissues at biopsy. Chronic infectious arthritis, particularly that caused by the tubercle bacillus, could not be considered in the light of the sterile fluid, lack of inflammatory reactions, and negative

results from guinea-pig injections. An atypical osteochondritis juvenilis of the tarsal scaphoid followed by its fragmentation, thereby creating a static imbalance in weight-bearing, was given credence when 2 painless cases of tarsal osteochondritis were found reported by Buchman.[1] But the x-ray plates made three weeks after injury show such extensive combined bone destruction and reparation at the astragaloscaphoid joint and in the scaphoid that in no way could the condition be classed as Koehler's disease. The finding that absence of pain with such a lesion is indicative but not pathognomonic of neuropathic joints made the situation more confusing. Steindler[2] reports 16 out of 99 neuroarthropathies which had varying degrees of pain. Milgram[3] also states that deep-tissue insensibility to needle puncture is not a constant finding in neuropathic joints, and that the occasional infection subsequent to biopsy is almost invariably accompanied by pain in that area.

Granting that the condition is typically neuropathic — which it apparently has been from the start to the present time — one is confronted with the necessity of explaining the neuropathogenesis. Mitchell[4] in 1831 first described such joint lesions, but it remained for Charcot[5] thirty-seven years later to make it a clinical entity by his complete description and his association of it with tabes dorsalis. During and immediately after the Civil War cognizance was given to the fact that traumatic injuries to the spinal cord or to nerve roots could produce exactly the same clinical and pathologic changes as those seen in a Charcot joint. To show the progress of this study since then and to apply it to this case, we give below the classification of neuropathic joints by Shands.[6] He places tabes dorsalis and syringomyelia in separate groups, as they are the causative factors in practically all cases.

1. Tabes dorsalis.
2. Syringomyelia.
3. Following lesions of the cerebrum:
   - Dementia paralytica.
   - Hemiplegia following hemorrhage.
4. Following lesions of the spinal cord:
   - Injury.
   - Congenital malformation.
   - Tumor.
   - Tuberculosis of spine.
   - Acute myelitis and anterior poliomyelitis.
   - Progressive (central) muscular atrophy, etc.
5. Following lesions of peripheral nerves:
   - Injury.
   - Peripheral neuritis.
   - Leprosy.

In this patient one can eliminate cerebral lesions, and with the exception of trauma, all spinal lesions. Complete spinal plates at two hospitals were interpreted as negative for either congenital or developed malformations. In considering the peripheral nerve lesions one may reject leprosy and peripheral neuritis by reason of the history and the clinical findings. There remain but the cord and peripheral lesions caused by trauma or by unknown or concealed factors. It is logical to consider only the peripheral nerve lesions, since the area of partial deep sensibility following trauma is sharply limited to the ankle and foot.

The first case of joint changes subsequent to peripheral nerve lesions was reported by Packard.[7] Mitchell[8] wrote of a patient who developed excessive joint changes after dislocation of the humerus. McArdle[9] mentions that arthritic changes may appear when a nerve to that joint has been pierced, rubbed, lacerated or otherwise subjected to trauma or irritation. It is known that atrophy of bone and soft tissue always occurs when a nerve is completely severed, but it is significant that neuropathic joints following pressure on the sciatic nerve by bone tumors have been reported. Wile and Butler[10] write of a patient who developed a typical neurotrophic joint following acute arthritis; no serologic or neurologic changes accounting for the condition were found. Shands[6] reports a neuropathic ankle which occurred several years after an injury to the lower leg; on dissection the distal portion of the posterior tibial nerve was found to be surrounded by cicatricial tissue. He concludes that the scar formation in some manner blocked the sensory fibers leading to the joint but left the motor branches unaffected. Bennett and Hinricson[11] note a case of a neurotrophic ankle from the joint fluid of which tubercle bacilli were isolated. They state that the sensory nerve endings must have been adversely affected, and that this left the joint structures without adequate protection. Cases following alcoholic neuritis have been reported, and others secondary to injuries to the posterior nerve roots. Duncan[12] records a painless swelling of a lumberman's wrist which appeared soon after his shoulder had been injured by a falling log. This patient also had sensory changes through his entire arm and shoulder, which indicated that the posterior roots or even the cord had been injured. Another common etiologic factor is trauma at or near the affected joint.

Cotton[13] tells of seeing several cases of so-called traumatic arthritis accompanied by swelling, and with slight to moderate pain, which upon arthrotomy revealed a joint cavity filled with detritus. This condition is in his opinion closely related to osteochondritis dissecans, but the histology and the macroscopic appearance of the loose bodies and the evident destructive and reparative processes in the joint indicate rather a neurotrophic condition. He states that after removal of the joint debris the pa-

tients who were treated by splinting, by full protection and later by physiotherapy made complete recoveries. Philips and Rosenheck[14] report in 2 patients three apparently neuropathic joints which appeared after localized trauma. In each case the usual progressive neuroarthropathy was found despite the absence of any positive blood, spinal fluid or neurologic changes. One patient, an ice carrier, sustained a severe strain of his shoulder. The man was dissatisfied with conservative treatment and went to another hospital, where a biopsy gave a negative report for tumor. Later his other shoulder showed a slight but similar affection, but both joints reached a quiescent stage and he was able to work without discomfort or mechanical handicap. He was observed for over two years. The other case was that of a lumberyard worker who had a history of trauma of the shoulder joint. He had a neuropathic joint filled with sterile fluid. He also responded to conservative treatment, and the swelling and disability gradually decreased over the ten months of observation. These authors point out that Virchow found and reported such joint changes following peripheral nerve lesions, but that he received no credit for this discovery. They conclude that the explanation may be the result of a certain type of injury upon a peculiarly constituted individual who is disposed to such a condition.

That the lesion is rare is shown by the report by Key[15] of 69 cases diagnosed as having neuropathic joints, all of which were proved to be definitely tabetic. Steindler[2] writes of 64 cases of which all but 2 were tabetic, these presumably being caused by syringomyelia. Campbell[16] says that he has seen many patients in whom the brachial plexus has been injured, severed or avulsed but that in no case did such a joint develop. Others state that they have never seen this syndrome with peripheral nerve injuries. Milgram[3] sums up the situation by asserting that if neuropathic joints do occur after such peripheral nerve trauma they must be extremely rare. According to him, analysis of most case reports shows that there have often been avulsions and tears of the cervical or dorsal roots, which might well be attended by associated injuries to the spinal cord. Philips and Rosenheck[14] state that the condition may be a new clinical entity, that it is apart from the usual neuropathic joint, and that it occurs more frequently than is generally believed. They emphasize the importance of its recognition in the diagnosis and treatment of industrial injuries.

Codman[17] agrees that in the patient here considered the neuropathy antedated the actual injury noted in the history and that thus the tarsal bone must have been so predisposed. The loss of the sensory afferent fibers allowed the joint to become indolent, vulnerable to trauma and unable to compensate in case of injury. As noted in the early x-rays by the increased density of the calcaneal epiphysis and by the amount of destruction and proliferation of the scaphoid, one must conclude that so extensive a condition would not have been produced in three weeks. There is also a marked increase in inorganic salts in the other tarsals. Thus, unnoted by the patient, an insensitive left ankle joint must have been in existence for several weeks or months before the recorded trauma was received. It is well known that increased fragility of bone occurs in a neuropathic joint, and that of the tarsal bones the scaphoid is the most frequently affected. This is because of its exposure to such heavy pressure and strains in ordinary walking. From this fracture and subsequent fragmentation of the scaphoid there resulted such an imbalance in the usual weight-bearing lines that a defensive reaction was produced. This increase in size of both the distal tibia and fibula is common in neuroarthropathies, and Potts[18] has given it the name of parosteal bone.

That the lesion was progressive is shown by the two x-ray plates, and that it was caused by the constant traumatization to an unprotected joint is shown by the marked improvement in the joint upon its being protected by the walking iron. That the neural mechanism is disturbed is evidenced by the slight pain present, which is not commensurate with what one would normally expect with such an extensive lesion. Eloesser[19] has produced similar neuroarthropathies in cats by traumatizing the joint after he had severed the sensory fibers leading to the legs. The joint fluid is caused by a traumatic synovitis, and the partially loose bodies in the joint are pressed-out osteophytes, forming islands of bone in varying stages of development in the synovial folds and on the exposed articular surfaces.

The x-ray plates were seen and the facts of this history discussed by the staffs of one Boston and two New York hospitals. The roentgenologists made a positive diagnosis of neuropathic joint; the neurologists were inclined to defer final diagnosis until confirming evidence of some known neurologic factor was presented; the pathologists were unwilling to make further comment, other than that the joint showed chronic synovitis, with concurrent bone destruction and repair; the orthopedists called the joint neuropathic, advocated treatment by adequate splinting and protection, but preferred to have the condition linked with some recognized neuropathy.

SUMMARY

1. A case of a typical neuropathic ankle joint, without demonstrable serologic or neurologic changes except partial insensibility of local deep tissues, is presented.

2. Such painless swelling following trauma is indicative but not pathognomonic of a neuro-arthropathy.

3. The condition was caused by an unknown neural lesion, probably peripheral and perhaps traumatic in origin.

4. The joint lesion resulted from repeated trauma to an insensitive unprotected joint.

5. Prognosis is uncertain until the primary cause is found.

6. The differential diagnosis of this presumably neuropathic joint condition and similar joint changes caused by known neuropathy is discussed.

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FIBROMA OF THE OVARY WITH ASCITES AND PLEURAL EFFUSION

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INTEREST in ovarian tumors has increased during recent years, and has necessitated a reclassification of ovarian embryology and pathology according to modern concepts. We should no longer speak of cancer of the ovary or sarcoma of the ovary, but must differentiate the type of carcinoma by its predominating cell structure and embryologic anlage. Primary tumors of the ovary are not uncommon and may be varied. This must be self-evident to anyone who reviews the complex structure of the ovary — an epithelial covering, the so-called germinal layer, a tunica albuginea, a thickened connective-tissue coat beneath the epithelium, a cortex containing the primordial follicles, a medulla containing a few tubular rests from the developmental anlages, and a connective tissue stroma reaching from the cortex to the hilum and attached to the mesentery or mesovarium. In addition, there are numerous and tortuous blood vessels, lymphatics extending from cortex to hilum through medulla, and nerves accompanying the ovarian artery. With this renewed interest in ovarian tumors there have been discovered in the body other pathologic conditions existing simultaneously with these tumors and apparently caused by them.

Presented before the New England Obstetrical and Gynecological Society, Hartford, Connecticut, April 28, 1937.

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We are particularly interested in a definite syndrome: ovarian tumor, with ascites and pleural effusion. To those unfamiliar with this combination it means malignancy. In Kelly's[1] *Gynecology* one finds this statement: "Fibroma is among the rarest of pelvic tumors, and is characterized by an indefinite multiplication of all the connective tissue elements of the ovary at the expense of the other histological components. The mass thus becomes a fibroid ovary which may further contain degeneration cysts, dilated blood spaces, and lymph spaces." In the first 10,000 gynecological admissions to the Johns Hopkins Hospital he recorded 10 cases with large fibroid ovaries and 3 with small calcified tumors, an incidence of 0.13 per cent. Ascites was frequently present.

Reviewing the English and American literature we find that ovarian fibromas comprise a small percentage of all ovarian tumors, ranging in different clinics from less than 1 to 4 per cent, and probably averaging 2 per cent. There are several reported cases where abdominal ascites was noted. Boldt[2] in 1910 reported a case of fibroid tumor of the ovary with ascites. In 1909 Mapes[3] reported a similar case, and presented a review of the literature as far back as 1840; in this review he found 71 cases of fibroma not quali-

fied by any notation of fluid in the abdomen or chest. In 1913 Titus,[4] of Boston, in discussing fibroma of the ovary, stated that ascites was an accompaniment in about half the cases. The following year Fullerton[5] discussed fibroid tumors of the ovary and commented on the frequent early occurrence of ascites. Hellman[6] in 1915 reviewed the literature and found abdominal ascites in at least 5 per cent of all cases of ovarian fibroma, distinctly less than Titus's 50 per cent. Hellman presented 2 cases of his own which showed ascites. Reel[7] in 1917 reported ascites in the presence of ovarian fibromas; Saint,[8] of Edinburgh, in 1919 also found ascites in the presence of a single ovarian fibroid. Richardson,[9] of London, presented a pathologic specimen of fibroma of the ovary which had been accompanied by ascites. In 1920 Clark and Gabe[10] reviewed the literature and noted the work of Coe in 1882 and of Hellman in 1915. They placed considerable diagnostic value on the presence of fluid in the abdominal cavity. In 1922 Macdonald[11] reported ascites accompanying ovarian fibromas.

Not until 1923 do we find any mention made of pleural effusion in the presence of ovarian fibroma. Owen[12] asserted that hydrothorax might occur with ascites. Hoon,[13] reporting from the Mayo Clinic, reviewed all their cases of ovarian fibroma from 1910 to 1921. Out of a total of 4175 tumors of the ovary, he found 55 cases in which the mass was removed at operation and which showed no other pathologic condition. There were in addition 94 fibromas associated with other lesions. Hoon ventured the surmise that the ascites present might be due to peritoneal irritation. Two of his patients also had bilateral hydrothorax, which disappeared after operation. No mention of pleural effusion was made by Likes[14] in 1935.

In the majority of textbooks ovarian fibromas are either ignored or mentioned only in passing. In Crossen's[15] *Diseases of Women* the author devotes only five lines to ovarian fibromas. He states that all solid tumors of the ovary are rare and comprise only about 5 per cent of all ovarian growths coming to operation. In the second edition of Curtis's[16] textbook, discussion of these tumors occupies but seven lines. This author states: "Less than 200 cases are recorded in the literature, but inquiry indicates that this is not a true index of their frequency." In Davis's[17] *Gynecology and Obstetrics,* one fourth of a page is given to fibromas along with fibromyomas and myomas of the ovary, and all are classified as relatively uncommon growths. Meigs,[18] in his excellent book *Tumors of the Female Pelvic Organs,* devotes four pages to this particular tumor.

Much credit is due to Meigs and Cass[19] for emphasizing the syndrome of ovarian fibroma, ascites and pleural effusion, for although it has been recognized that ascites exists very frequently with fibromas of the ovaries, only very recently has the presence of hydrothorax accompanying these tumors been detected. In 1936 they reported a series of 7 cases of fibroma of a single ovary with fluid in the abdomen and in the chest. Four of these were from the Massachusetts General Hospital, 2 from the Mayo Clinic and 1 from the literature.

In all these cases the surgical removal of the fibroma proved to be the only step necessary to relieve the patients of ascites and pleural effusion. The reason for the fluid was neither known nor explained, nor was it at all clear why the removal of the tumor cured the condition. One ovary, never both, was involved, and the right chest was more often involved than was the left. Guinea-pig inoculations with the chest fluid were negative for tuberculosis, and cardiorenal disease was absent. Simple removal of the tumor produced a cure. Thoracic and abdominal paracenteses did not relieve the condition in the least. The entity was found to progress rapidly up to the time of operation.

We wish to report 2 cases, one a unilateral fibroma, the other bilateral and containing a papillary cyst in one ovary, in addition to the fibroma.

*Case 1.** E. B., aged 50, a white, married female, was admitted to the gynecological service of the Hartford Hospital on September 15, 1936, complaining of progressive enlargement of the abdomen of 6 weeks' duration, and of difficulty in voiding for a relatively short time. During the previous 6 weeks she had experienced lower abdominal pain, characterized as a heavy, dragging sensation. The former history was unimportant. Menopause occurred 10 years prior to admission, and since then there had been no bleeding.

The positive findings on physical examination were a general appearance of illness; two lipomas on the back; signs of fluid in the right posterior chest; slight enlargement of the heart to the left; an elevated pulse rate; and a pelvic mass rising to the umbilicus, which gave the findings compatible with encapsulated fluid. Roentgen examination of the chest was reported as follows: "Stereoscopic and lateral examination of the thorax shows a moderately large amount of fluid in the right pleural cavity, extending into the interlobar fissures and apparently encapsulated in the axilla. The possibility of an underlying pathologic change in the right pulmonary field cannot be excluded without further examination made after a thoracentesis. The left lung is negative in appearance."

An operation was performed under spinal anesthesia 3 days after admission. A lower midline incision was made. The abdomen was filled with blood-tinged fluid. A solid right ovarian tumor, with loops of bowel adherent to the tumor mass, was visualized. The liver was smooth, and normal to palpation. The gall bladder was negative.

*Reported through the kindness of Dr. G. E. Howe, on whose service at the Hartford Hospital the case occurred.

There was no sign of malignant metastases. The uterus, left tube and ovary appeared normal. Only the tumor was removed.

The pathological report (Dr. Kendall) was as follows: "*Gross.* Nodular mass 15 by 12 by 11 cm., encapsulated. On section, it is composed of interlacing strands of rather soft fibrous tissue, through which there are extensive interstitial hemorrhages and dilated vessels. Over one surface is what appears to be a vessel containing blood clot. At one pole of the nodule there is a cyst 3 cm. in diameter, having a ragged, seminecrotic lining. *Microscopic.* Multiple sections through the nodule show a rather cellular fibrous-tissue proliferation. There are some mitotic figures; a few are not entirely regular. There are extensive areas of myxomatous degeneration, and large vascular spaces are scattered through the tissue, in some of which thrombi are present. Structure is fairly characteristic of a benign fibroma of ovarian stroma. *Diagnosis.* Fibroma of ovary."

The patient was discharged on the 19th postoperative day, after an uneventful convalescence. An x-ray of the chest at that time showed less fluid than prior to operation. An x-ray of the chest 7 months after operation showed no evidence of effusion.

Case 2. I. U., aged 55, a white female, was admitted to the gynecological service of the Municipal Hospital, Hartford, on August 21, 1936, complaining of swelling of the abdomen. About 2 months prior to admission she

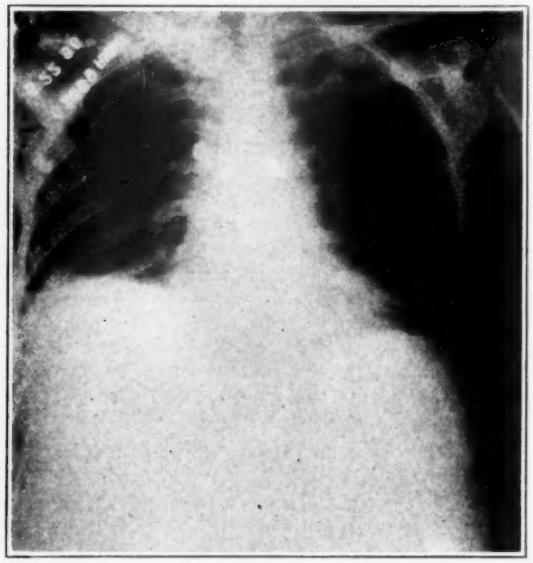

Figure 1. *Roentgenogram of chest before operation, showing pleural effusion.*

first noticed the swelling. She also complained of pain in both kidney regions and a low sacral backache. There was a history of chronic constipation. The menopause had occurred at 35. The past history was unimportant, except for hospitalizations for glaucoma of the eye and for strangulated hernia. Examination revealed bilateral cataracts; a few coarse rales at the right lung base, with diminished breath sounds over the same area; a blood pressure of 112/68; the abdomen shiny and taut with presence of a fluid wave; and a right inguinal hernia in addition to the old herniorrhaphy scar in the same region. Vaginal examination showed a large, hard, irregular mass filling the pelvis, reaching to 2 fingers below the umbilicus, immovable, with tenderness in the region of the sigmoid and rectum. The mass filled the posterior cul-de-sac.

X-ray of the chest the day after operation showed no evidence of tuberculosis, but there was increased density at the outer and lower portions of the right lower lobe, with irregularities of the diaphragm and partial obliteration of the costophrenic angle, all indicative of pleural exudate (Fig. 1). The blood Wassermann and Kahn tests

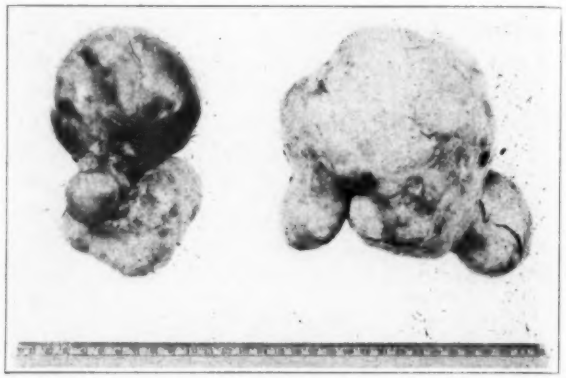

Figure 2. *Fibromyomas of both ovaries; papillary cyst of ovary.*

gave a 2+ reaction. The blood nonprotein nitrogen was 35.9 mg. per cent. The red-blood-cell count was 4,030,000 with 85 per cent hemoglobin.

At operation about 3500 cc. of straw-colored fluid was removed from the peritoneal cavity by suction. A solid ovarian tumor 9 cm. in diameter, together with a multilocular cyst, was removed. The lower pelvis was then found to be filled with another ovarian tumor, so situated as to give rise to sigmoidal and rectal obstruction; it was

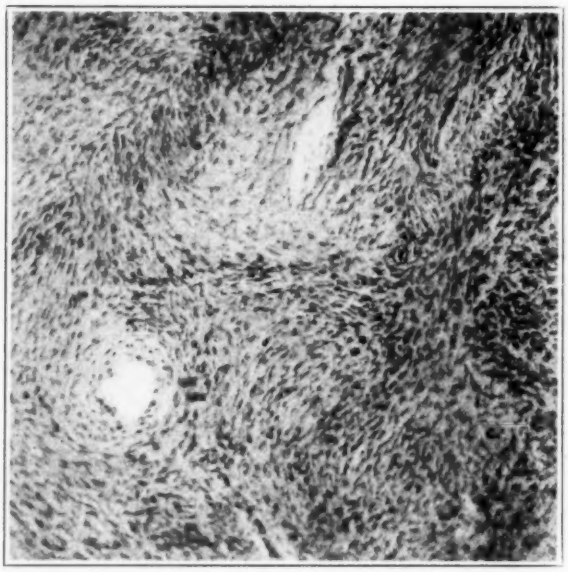

Figure 3. *Photomicrograph of ovarian fibroma.*

delivered from the pelvis with some difficulty and was removed. Both tubes and the uterus down to the cervix were then removed by the usual procedure followed by a supracervical hysterectomy. No lymph nodes were found

in any part of the peritoneal cavity, nor was there any evidence of extension of the tumor masses to the bowel. There were no bowel adhesions. Each tumor apparently arose from an ovary (Fig. 2). Grossly the tumors appeared smooth and nodular; on section they were composed of dense gray strands of fibrous tissue. Microscopic sections of the solid tumors showed typical fibromas, no anaplasia and no malignancy (Fig. 3). In one or two areas the pathologist reported the cells to be somewhat large and slightly hyperplastic. The cyst was lined by low epithelium, which in focal areas was thrown into a papillary arrangement. Here again there was no evidence of malignancy.

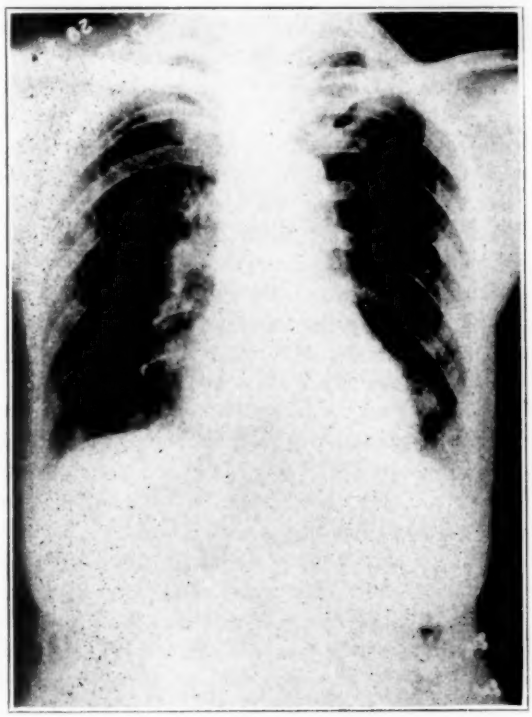

Figure 4. *Normal roentgenogram of chest three months after operation.*

The patient had an uneventful convalescence and left the hospital on the 17th postoperative day. X-ray examination of the chest taken November 30, 3 months after operation, "showed no evidence of pleural effusion" (Fig. 4).

SUMMARY AND CONCLUSIONS

There is a definite syndrome of ovarian fibroma, ascites and pleural effusion which has only very recently been recognized. Two such cases are described. The cause of the fluid and the reason why removal of the ovarian tumor effects a cure remain unexplained. Many of these cases may be found on medical wards. Internists considering causes of chest fluid and ascites should be aware of the condition, as should surgeons and gynecologists considering the possibility of a malignant tumor with metastases to the chest. Usually but one ovary is involved, but both may be, as noted in a case reported herewith. The cure is surgical removal of the tumor. Irradiation therapy is unnecessary.

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CASE RECORDS OF THE MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 24061

PRESENTATION OF CASE

First Admission: A seventy-one-year-old, white toolmaker entered the hospital with the complaint of gradually increasing dyspnea of eighteen years' duration.

About twenty-five years before entry he had an attack of bronchitis followed by "blood poisoning" in his hand which remained localized. Shortly after that he noticed occasional swelling of his ankles, especially when he stood for a long time. Eighteen years before entry he began to notice dyspnea on exertion and more frequent swelling of his ankles. The swelling always disappeared during the night. During the next thirteen years the symptoms gradually became more marked, and he had recurring head colds and attacks of bronchitis. About five years before entry he began to use two pillows at night. He became dyspneic on moderate exertion, and his ankles were swollen every night. Two years before entry he slowly gained about 26 lb. until his weight reached 220. He said that there was no increase in his appetite or change in his eating habits. One year before entry he had a severe attack of bronchitis, and for the first time it was necessary for him to stop work. At that time the edema of his legs had become quite marked, and he developed ulcerations of his lower legs. He remained at home for seven weeks, and then returned to work. His dyspnea had become so marked that he had considerable trouble climbing one flight of stairs, and he had an almost continuous cough, productive of small amounts of mucoid sputum. Six months before entry he noticed edema of his scrotum and slight swelling of his abdomen. Two months later he had another attack of bronchitis with paroxysms of coughing and questionable fever. He stayed in bed for two weeks because of weakness, and after that was unable to return to work. His physician gave him digitalis, which failed to bring about much improvement. During the four months before entry he had nightly attacks of "asthma" which lasted for about half an hour and subsided spontaneously. On several occasions it was necessary for him to spend the night in a chair in order to get any sleep.

One sister died of "dropsy" and one had "dropsy" at the time the patient entered the hospital. His family history was otherwise negative, and his past history was noncontributory.

Physical examination revealed a well-developed man breathing rapidly, with cyanosis of his lips and nailbeds. The lungs showed dulness to flatness at the right base with absent breath sounds, voice sounds and tactile fremitus, and a few fine moist rales at the left base. The heart showed general enlargement with the left border of dulness 13 cm. from the midline. The sounds were of poor quality, and A_2 was equal to P_2. There was a loud systolic murmur at the apex, transmitted to the sternum and axilla. The blood pressure was 140 systolic, 80 diastolic. There was obvious fluid in the abdominal cavity. The subcutaneous tissue of the abdomen, genitalia and legs was edematous.

The temperature was 98.6°F., the pulse 80. The respirations were 25.

The urine had a specific gravity of 1.025 and contained rare hyaline and granular casts. The blood showed a red-cell count of 5,250,000 with 80 per cent hemoglobin, and a white-cell count of 7600, 59 per cent polymorphonuclears. The stool examination was negative. The blood Hinton test was negative. The nonprotein nitrogen of the blood was 28 mg., and the protein 5.9 gm. per cent. The sedimentation rate was 0.52 mm. per minute.

An electrocardiogram showed a normal rhythm, inverted T_1 and T_2, low and upright T_3, and upright T_4. Q_4 was present, S_1 prominent. The S-T intervals in Leads 1 and 2 were depressed. There was a slight tendency to low voltage and right-axis deviation.

He was treated with large doses of digitalis and with salyrgan, and in three weeks all evidence of cardiac decompensation had disappeared. There was still moderate edema of the legs, particularly on the left; but this was thought to be due to varicose veins or an old phlebitis. He was discharged on the thirty-second day on a digitalis and theobromine regime with Bender bandages for his legs.

Final Admission (six weeks later). After discharge he continued to be dyspneic even on slight exertion, and soon noticed that in spite of exercises, bandages and elevation of his legs the edema began to reappear. His physician took him off digitalis and gave him another medicine which he said contained digitalis. During the two weeks before re-entry he became very orthopneic, the edema spread up to his hips, and he felt very weak and tired.

Physical examination revealed a dyspneic, cy-

anotic man propped up in bed, in obvious distress. There were moderate dulness and moist rales at both lung bases. The heart was essentially the same as on previous examination, and the blood pressure was 140 systolic, 80 diastolic. The abdomen was distended with fluid, liver and splenic dulness were increased, and there was marked edema of the legs, sacrum, scrotum and hands.

The temperature was 99.4°F., the pulse 84. The respirations were 36.

The urine contained a trace of albumin. The white-cell count of the blood was 11,000. The blood nonprotein nitrogen was 37 mg., and the protein 4.9 gm. per cent.

An electrocardiogram taken on the fifth day showed a rate of 133 with sinoauricular tachycardia, depressed S-T_1 and S-T_2 intervals, inverted T_1, low diphasic T_2, practically flat T_3 and flat T_4. Q_3 was present, Q_4 short. The P waves were poorly marked, and S_1 was prominent. The QRS complexes were slightly slurred, and there was slight right-axis deviation.

In spite of digitalis, ammonium chloride and salyrgan therapy he failed to show any improvement. He died on the sixth day.

Differential Diagnosis

Dr. James H. Means: This is a rather unusual story of rapidly decreasing cardiac reserve over an eighteen-year period in a man who was seventy-one years of age at the time he came under observation. We observe these symptoms of increasing dyspnea related to effort and the gradual development of orthopnea. We note edema which is clearly present at first only when he is upright, disappearing at night, and finally we are told of the development of a considerable degree of generalized cardiac edema which persists and gradually ascends his legs. It seems to me that the edema in the early part of this story is rather more than one would expect solely on the basis of cardiac insufficiency. We know he had some ulcers on the lower legs and are therefore inclined to think there was another factor, namely varicose veins, with secondary varicose ulcers. The cardiac patient with varicosities will get edema more quickly than one who has no varicosities. I am impressed with the story of repeated attacks of bronchitis. He has them over and over again. We learn that he has paroxysms of coughing, possibly fever, and finally we are told that he has attacks which resemble asthma; so we are justified under the circumstances in supposing they are so-called cardiac asthma, and a manifestation of heart failure. On the historical data alone one would be entitled to say that he has cardiac failure which involves both sides of the heart.

When we come to the physical examination, we are told that he is well developed — indeed we are told that he has gained 26 lb., bringing his weight up to 220, so he was an obese subject anyway. We do not know the cause for this gain in weight. It may have been dropsy, but it occurred at a time when he was still able to get about. It is unusual for a man to gain 26 lb. from dropsy and still be able to continue with work; but it is possible.

Here is a man then with a large heart, with a systolic but no diastolic murmur. He is described as being short of breath and having cyanosis. No mention is made of the size of the liver, and there is nothing about dilated veins in his neck, so we may assume that the liver is not enlarged and that the veins are not dilated. He apparently has a right hydrothorax and some edema in his left lung.

He is afebrile; from the urinary and blood findings there is no evidence of any disease. He has no azotemia. The protein of the blood is within normal limits. The sedimentation rate is a little high. All I can get out of the electrocardiogram is right-axis deviation. For further interpretation I shall be obliged to depend on the cardiologist. Perhaps someone will speak about that later.

I might say that the laboratory and physical findings check with the history and indicate that this man is suffering from some kind of heart disease, with congestive failure and consequent anasarca. His response to digitalis is further evidence that he has congestive heart failure. It is interesting that the pulse rate is the same on his second entry as on the previous entry. Although we do not know whether he was digitalized on the first entry, we assume that he was not. This time he is, but the pulse rate is identical. The respirations are more rapid than previously.

No mention is made of x-ray studies.

Dr. Tracy B. Mallory: None were made.

Dr. Means: Might I have the help of the cardiologist on the electrocardiograms if he does not tell me the diagnosis?

Dr. Mallory: I think that would be in order.

Dr. Edward Bland: The first electrocardiogram is consistent with a man of his age, except the right-axis deviation. The T waves are suggestive of a digitalis effect. We do not know whether he was getting it at the time of the first entry. The second electrocardiogram showed a rapid rate, depressed ST_1 and ST_2 and probably represents a digitalis effect. A flat T_4 also suggests digitalis. Here again he had slight right-axis deviation without definite intraventricular block. That suggests a little more than the normal findings in a man of seventy-one.

Dr. Means: It seems obvious that he died of congestive heart failure. The only question is, What kind did he have? The way to approach

such a question is to consider all the kinds he could have had, and by elimination, see if one can arrive at a correct diagnosis. Did he have rheumatic heart disease? I suppose it is possible. I have a patient, a man of his age, who has rheumatic mitral stenosis, so one can live that long with mitral heart disease. By some remote possibility he might present a picture like this one; but it is extremely unlikely. There is nothing in the previous story to suggest that he had had heart disease prior to this eighteen-year story. The whole course seems unlikely for rheumatic heart disease. The physical findings of a systolic murmur and a heart transversely widened would be consistent with organic mitral regurgitation, but we have no evidence of stenosis, and altogether the picture does not closely resemble rheumatic heart disease. Syphilis, so far as I know, does not run this long. He is too old for it and certainly has had symptoms too long for syphilitic heart disease. As he also had a negative Hinton test, I shall rule out syphilis, although we must recognize that a few patients with negative serologic tests do have syphilitic heart disease. There is no evidence of hypertensive heart disease, and no evidence in the history, physical examination or electrocardiogram that there is anything whatever wrong with his coronary arteries. There is no evidence of myxedema or thyrotoxicosis.

So by exclusion we are almost led to believe that his heart disease must be secondary to some lesion in the lesser circulation and that this is indeed a case of cor pulmonale. It seems to me that this is suggested by several things. In the first place, cyanosis is mentioned, and cyanosis is more striking in older people with pulmonary heart disease, as I have seen them, than it is in people with other types. It is also suggested by right-axis deviation, although we must concede that organic mitral disease may give a similar picture under certain circumstances. Nothing is said in the physical examination about emphysema, but I am very much impressed with this story of repeated episodes of what is called "bronchitis." It seems to me that coincident with this long history of very gradually progressive heart failure something has been going on in the lungs that was called bronchitis and it may have been associated with a certain degree of gradually increasing pulmonary emphysema. The attacks which he had had were called "asthma," and they would fit into such a conception, although of course they might also have been due simply to acute pulmonary edema as a result of left ventricular failure. I do not believe it is possible to say which they were without having the opportunity to observe them.

I do not believe that he has Ayerza's disease, — he is far too old, — but there are other things that could go wrong with the pulmonary circuit.

I am reminded very much of a case that I had in the Phillips House a number of years ago that Dr. Mallory did an autopsy on and that we reported together. The man was not so old as this one. He came in with heart failure. We never could figure out why he had so much cyanosis. There was a considerable degree, with nothing apparent in the lungs to explain it. It turned out at autopsy that he had a complete thrombotic occlusion of one pulmonary artery with a collateral circulation through the bronchial artery, which was as big as my little finger. It was a chronic closure of one pulmonary artery, and the compensatory circulation had taken care of the nutrition of the lung. The lung was therefore all right, indeed it was in better shape than the lung that was not shut off from the pulmonary artery because the latter had edema while the former did not. Air went in and out, so that it gave normal physical signs. That case taught me that when you have a cyanosis that seems to be a little difficult to explain you should think of some process in the pulmonary circuit, possibly a thrombosis or perhaps an embolic process. I think Dr. Mallory has taught us of late years that multiple infarcts in the lungs are not at all uncommon. We find over and over again that people have showers of small infarcts in the lungs; and something of that kind may have taken place in this case, so that one might have a combination of simple cardiac failure and some such process in the lungs aggravating the condition. I wonder if the episode that occurred just before the second entry, when he became orthopneic and had rapid spread of edema, might not have been due to something of that sort. I can only guess at that because I have none of the actual criteria to make such a diagnosis. I have no story of bloody sputum — nothing that is definite proof — no story or description of engorged neck veins. I suspect, however, that multiple pulmonary infarction is a good possibility.

The best I can do with this case is to say that the man did have congestive heart failure with a large heart. I should expect to find both sides hypertrophied and dilated, and to find edema of the lungs, hydrothorax and ascites. We know he had all these things. I suspect that something will be found in the pulmonary artery in the shape of thrombosis with multiple infarcts. I am intrigued with the story about the veins in the legs and wonder if the situation is a composite one of congestive heart failure due to chronic pulmonary disease and on top of that a phlebitis related to the varicosities in the leg that may provide a source for emboli. That is all I can do with the data I have. I suppose I have forgotten some-

thing or failed to appreciate the significance of some fact given in this record — we usually do that. There are various fantastic diagnoses that one could indulge in. Dr. Mallory once produced a tumor of the heart that gave congestive failure, but I will not enter into any such speculation as that.

DR. MALLORY: I should like to raise the question of senile calcified aortic stenosis.

DR. MEANS: I suppose I should have thought of that, but there is no mention of a murmur that in the slightest degree suggests aortic stenosis. I cannot make a diagnosis of aortic stenosis without a basal systolic murmur and thrill.

DR. MALLORY: He had a systolic murmur.

DR. MEANS: It was at the apex and transmitted to the axilla.

DR. MALLORY: I mentioned that merely because of all the valvular heart disease we see in this hospital that is the one most frequently missed.

DR. MEANS: Is that what he had?

DR. MALLORY: No.

DR. JOHN H. TALBOTT: I should like to ask a cardiologist if it is possible for a congenital pulmonary stenosis to be asymptomatic until the man was well along in years.

DR. MEANS: I did not mention congenital lesions. Dr. White had one patient who died of congenital heart disease at an advanced age but he was not so old as this patient.

DR. MALLORY: He was fifty-five, but had had symptoms for many years.

CLINICAL DIAGNOSES

Arteriosclerotic heart disease with congestive failure.
Pulmonary infarct?
Anasarca.
Varicose veins.

DR. MEANS'S DIAGNOSES

Pulmonary heart disease (cor pulmonale).
Multiple pulmonary thrombosis.
Congestive heart failure.
General anasarca.
Varicose veins.
Phlebitis with resulting pulmonary embolism?

ANATOMICAL DIAGNOSES

Embolism and thrombosis, old and fresh, of many pulmonary-artery branches.
Pulmonary infarcts, recent.
Cor pulmonale.
Anasarca.
Pulmonary tuberculosis, bilateral, healed.
Arteriosclerosis: marked, abdominal aorta, iliac and popliteal arteries; moderate, coronary arteries.
Thrombophlebitis: old, popliteal veins; recent, inferior vena cava.
Benign hypertrophy of prostate.
Duodenal ulcer.
Diverticula of colon.
Rectal polyp.

PATHOLOGICAL DISCUSSION

DR. MALLORY: I thought it would be interesting to give Dr. Means a test and see if he remembered the case on which he based a paper about ten years ago. This is the most similar case that we have had since. He has presented it so skilfully that he has made it seem easy and obvious, but I should like to call your attention to the fact that cor pulmonale was not even mentioned as a possibility during either of this man's two sojourns in the hospital. This man, instead of having a single huge thrombus obstructing the main pulmonary artery to one lung, had multiple old organized thrombi in the smaller subdivisions of the arterial tree. One of the major branches to the right upper lobe, the major branch to the right lower lobe and the main branch to the left lower lobe were all occluded by old organized and partially recanalized thrombi. In this case there was no compensatory dilatation of the bronchial artery that we made out in gross. There probably was some that could have been demonstrated if anyone had guessed the diagnosis before autopsy as accurately as Dr. Means has and had injected the lungs with proper technic. In this case I do not believe it would have been very great, however, because there was fairly extensive recanalization of most of the thrombi, and a very marked dilatation at the vasa vasorum which anastomosed extensively with the canalizing vessels. In one of the large canalizing vessels within one of the biggest of the old thrombi, we found a fresh thrombus, which I think may be correlated with the acute episode just before he came in the last time. We also found a number of small recent infarcts way out in peripheral portions of the lung. His popliteal veins showed an occlusive process of an age at least equal to that of the older pulmonary lesions. Both had obviously been at one time completely occluded and had become fairly extensively recanalized. In the inferior vena cava we found a fresh thrombus which was probably the source of the final pulmonary emboli. The lungs, except for the vascular change, were in remarkably good condition. There was no evidence of bronchitis or of emphysema. There was some atelectasis as the result of the bilateral pleural effusions and the apices showed old tuberculous scars.

He had a variety of other conditions, none of them apparently of any clinical importance, such

as a duodenal ulcer, a rectal polyp, diverticula of the colon and a hypertrophied prostate.

A PHYSICIAN: Did he have any disease of the mitral or aortic valve?

DR. MALLORY: No. He had slightly calcified atheromatous plaques on the posterior surface of the mitral valve, a very common spot for them at that age, and they never, so far as I know, have any functional significance. The heart weighed 525 gm. The right ventricular wall measured 8 mm. in thickness, and the columnae carneae on the right were larger than those on the left. The pulmonary orifice was normal in circumference but the tricuspid valve measured 16.5 cm., which indicates considerable dilatation. The coronaries showed some calcification but no significant narrowing. The liver showed a slight degree of cardiac cirrhosis.

DR. MEANS: The loud systolic murmur was probably functional.

DR. MALLORY: I should think it must have been.

CASE 24062

PRESENTATION OF CASE

First Admission. A sixty-year-old Jewish housewife entered the hospital with the complaint of abdominal pain of two and a half days' duration.

Two and a half days before entry the patient was quite suddenly taken with a severe pain in the epigastrium which seemed to start on both sides and constrict toward the middle, with radiation through to the back, but not to the shoulders, arms or substernal region. The pain was not related to exertion, emotion or the intake of food. It was accompanied by nausea, perspiration, hot and cold feelings, some faintness and dizziness, but no vomiting or chills. She went to bed, but the pain continued unrelieved by aspirin until the time of entry. She was unable to sleep, and in spite of various laxatives failed to have a bowel movement.

For the previous three years she had had occasional similar attacks of pain which were very mild and of brief duration. During that period she had noticed gradually increasing anorexia so that her diet became very limited, although she still remained very obese. Her greatest weight had been 200 lb., and at the time of entry she weighed 180 lb. She thought she had lost about 10 lb. during the week before entry. During the year before entry she had become constipated. Previously she had never had any trouble with her bowels, but it became necessary for her to take large doses of laxatives to obtain a movement. She stated that her stools had been black during this period but never bloody or clay colored. She had never had any jaundice or other gastrointestinal symptoms.

She had had malaria as a child but denied ever having had typhoid or venereal disease. She experienced some dyspnea on climbing stairs, and she said that her ankles had been swollen for years. She had no other cardiorespiratory symptoms. For two years she had had some nocturia and frequency. The family history was noncontributory.

Physical examination revealed an extremely obese woman in considerable discomfort from abdominal pain. There was a rough systolic murmur heard all over the precordium, and the heart sounds were of good quality. The blood pressure was 150 systolic, 100 diastolic. The abdomen showed tenderness to deep palpation in both upper quadrants, more marked on the right just below the costal margin. There were no masses or spasm. One observer thought the liver edge was palpable. There were bilateral varicose veins with varicose ulcers on both lower legs.

The temperature was 100.5°F., the pulse 90. The respirations were 20.

The urine examination was negative. The white-cell count of the blood was 7900. The hemoglobin was 85 per cent. The nonprotein nitrogen of the blood was 27 mg. per cent, and the van den Bergh was slightly above normal, with an indirect reaction. A blood Hinton test was negative.

A Graham test showed normal filling and emptying of the gall bladder, and a gastrointestinal series was negative.

She vomited once shortly after entry, and on the second hospital day had a shaking chill, although her temperature had fallen to normal. On the ninth day a barium enema showed no evidence of a pathologic lesion, and on the following day the Graham test was again negative. The pain subsided during her stay in the hospital, and she was discharged on the eleventh day.

Final Admission (eight months later). The patient was fairly well for about two months following discharge, but then began to have severe, constricting pain deep in the right upper quadrant, which did not radiate. The pain was constant, unremitting and crushing in nature, not sharp or colicky. She had had no known fever or jaundice, and believed that there had been no change in the color of her stools or urine. On one occasion, a short time before re-entry, she had a shaking chill. She developed severe anorexia and, when she did eat, felt nauseated but never actually vomited. About a month before re-entry she went to bed because of weakness and continued pain and remained there until she was brought to the hospital. Two weeks previously she developed dyspnea which was not relieved by sitting up and

was not accompanied by cough or other symptoms. By that time the pain had become very severe and had spread to the left side, deep in the upper quadrant.

Physical examination revealed an extremely obese, acutely ill woman lying listlessly in bed. The skin was sallow. There was marked tenderness in the mid-epigastrium, becoming less acute laterally along the costal margins. A smooth, firm, movable, non-tender mass with rounded edges and smooth surfaces was palpable in the left flank and extended almost into the pelvis. The heart and lungs were negative except for the systolic murmur previously noted. The blood pressure was 100 systolic, 70 diastolic.

The temperature was 99.5°F., the pulse 110. The respirations were 30.

The urine examination was negative. The blood showed a red-cell count of 3,570,000 with 70 per cent hemoglobin. The white-cell count was 10,200, 57 per cent polymorphonuclears and no abnormal forms in the smear. The nonprotein nitrogen of the blood was 59 mg. per cent, the protein 4.2 gm. The van den Bergh was normal, indirect. A blood Wassermann test was negative.

An x-ray of the chest showed the right diaphragm to be high and flattened. There were several dense areas just above the right diaphragm which appeared to be areas of atelectasis. The upper portion of the right lung and the entire left lung were clear. A flat plate of the abdomen showed the kidney outlines to be distinct. The left kidney was not quite so low as the right, and there were no stones. A large soft-tissue mass was present in the left upper quadrant which extended down to the region of the crest of the ilium and had the general shape of the spleen. The shadow of the liver did not seem to be grossly enlarged. An intravenous pyelogram showed poor excretion by both kidneys. The pelves and calices were not seen, and the bladder was not filled. A Graham test was done and failed to show any gall-bladder shadow. No gallstones were demonstrated. A barium enema was negative.

On the third day her skin seemed to have a somewhat indefinite, bronzed color and the blood pressure was 82 systolic, 64 diastolic. She was apparently failing rapidly. There were questionable signs of fluid at the right lung base. A consultant reviewed the blood smear and was of the opinion that it showed about 4 per cent very immature white cells. On the seventh day she was very dyspneic, and her extremities were cold, clammy and cyanotic. The nonprotein nitrogen was 130 mg. and the carbon-dioxide combining power was 28.8 vol. per cent. The serum sodium was equivalent to 110.7 cc., and the serum chloride to 88 cc. of N/10 sodium chloride. She died that day.

Differential Diagnosis

Dr. Richard B. King: It seems to me that the decision we have to make here is whether the first admission had anything to do with her death. I am inclined to believe that she had two unrelated conditions and that the first admission was in some way related to the gall bladder; that is what they were thinking about of course. The description of the attack is perfectly consistent with gall-bladder disease. It lasted two and a half days—a very unusual duration for pain associated with diaphragmatic hernia or acute ulcer. The fact that they mention it was not related to exertion or motion indicates that they thought of coronary thrombosis, but did not believe it was that because they did not have electrocardiograms made. The blood pressure was 150 systolic, 100 diastolic, and examination of the heart itself failed to reveal anything noteworthy. The description of that attack is perfectly consistent with gallstones or cholecystitis, and probably not related to the thing that killed her. It is interesting that she stated that the stools had been black, off and on I presume, for a year. It is always questionable how much importance you can put on such a statement without knowing whether she had taken an iron tonic which would have colored the stools. There was no evidence of trouble in the gastrointestinal tract at the time of first entry. She had a normal gastrointestinal series and a negative barium enema. They were looking for gall-bladder disease, and they did a second Graham test because they were skeptical about the first. We all know, however, that one can have gall-bladder disease without its showing in the Graham test. The shaking chill is curious; I should like to know whether she sweat following it and whether the temperature, which had fallen before the chill, went up again. I do not know how to interpret it. I should guess that at the time of the first admission she had gall-bladder disease, with a long attack of gall-bladder pain.

When we come to the second admission we realize that a lot has happened in the interim, and it is this story which concerns us in regard to the patient's death. The pain in the right upper quadrant, which was constant, unremitting and crushing, is hard for me to believe. I do not see how a patient could live for six months with unremitting, crushing pain, night and day. It seems to me that if it was as severe as described she would have been unable to sleep and would have died of exhaustion. It is impossible for me to relate the pain with the gall bladder, and I do not know any condition in the liver which could cause pain

of that sort and which would last six months. It might be a nerve pain in the sense that one can have shingles with pain lasting for years, and I should be inclined to interpret it as having something to do with pressure on, or infiltration of, nerves. She was then getting short of breath and in the habit of sitting up. She was probably developing anemia. None of these things suggest trouble in the cardiovascular system. The original swelling of the feet and the shortness of breath which she had had for years could be explained on the basis of the varicose veins and obesity.

The whole crux of this matter seems to rest on whether the mass felt on the physical examination was the spleen. All the evidence points to its being spleen. They did everything to rule out the kidney. It was not a tumor related to the kidney, and a tumor arising from the pelvis would not give that kind of shadow. They made no note of a notch; this is disturbing because in spleens of that size we almost always feel a notch and describe it. I am convinced, however, that the mass was the spleen. Assuming this to be so, we have to explain the anemia, the finding of queer cells in the blood smear, the pain, the patient's death. It seems to me that a lot happened more or less simultaneously in relatively widely separated areas, — the rise in nonprotein nitrogen, the fall in blood pressure, the low serum sodium, — which suggests that we are dealing either with metastases to various places or a widespread vascular disorder, or with what seems more likely to me leukemic infiltration of various organs. As to the cause of death, it could have been renal or adrenal failure, or a combination of the two. The findings in the chest could be leukemic infiltration or irritation of the pleura or lungs, or both. I believe that the patient had gall-bladder disease, not related to death, and aleukemic leukemia, type undetermined. There is no description of these peculiar cells which were seen in the blood smear, and the fact that they were not named makes me feel that the observer was unable to classify them.

Clinical Discussion

Dr. J. W. Zeller: When I saw this patient a few days before death, I thought the mass in the left upper quadrant was the spleen although it was disturbing that it had not been noticed there eight months previously. The lymph nodes were certainly not striking. The inguinal and axillary nodes were not palpable, although at autopsy they looked as if they should have been, in spite of her obesity. The blood smear showed a few young cells; I did not know exactly into what series they fell but thought they were lymphocytes. I thought she had lymphoblastoma. The systolic murmur was very harsh and certainly indicated some organic disease of the mitral valve.

Clinical Diagnoses

Coronary thrombosis ?
Chronic cholecystitis ?
Lymphoblastoma ?
Arteriosclerotic heart disease.

Dr. King's Diagnoses

Chronic cholecystitis.
Aleukemic leukemia, ? type.
Leukemic infiltration of kidneys, adrenal glands, spleen, lungs and retroperitoneal lymph nodes.

Anatomical Diagnoses

Aleukemic lymphatic leukemia.
Thrombosis of iliac and pelvic veins and inferior vena cava.
Edema of the legs.
Hydrothorax, right.
Arteriosclerosis: slight, coronary; moderate, aortic; moderate, mitral and aortic valves.
Nephritis, chronic vascular.
Obesity.

Pathological Discussion

Dr. Tracy B. Mallory: The autopsy showed a very large spleen that weighed over 1200 gm. and a diffuse enlargement of lymph nodes all over the body, particularly the retroperitoneal nodes. The liver was normal in size, but the markings were a little exaggerated — the branchings of the portal tree could be seen with a distinctness that you do not see in a normal liver but do at times in leukemic infiltration. The gall bladder and bile ducts were absolutely negative. The heart showed partially calcified, atheromatous plaques on the mitral valve; the valve margins were normal, and I do not believe its function was interfered with. The one other important finding in gross was a very extensive thrombophlebitis of both iliac veins and of the deep pelvic veins, which had extended up the vena cava as far as the mouth of the renal vein. It was not a completely occluding thrombus, however, filling only about half the lumen. Microscopic examination showed a typical leukemic infiltration of the spleen, lymph nodes, liver and bone marrow. It was of undifferentiated type, but probably lymphoid.

A Physician: What about the adrenals?

Dr. Mallory: They showed microscopically a little lymphoid infiltration but nothing in gross and nothing that I think could have interfered with function. The kidneys showed a moderate

grade of vascular nephritis, not enough, however, in itself to explain uremia. I should be inclined to attribute the renal failure to a combination of factors in a moribund patient. She was hypertensive when she first came in so that she had kidneys accustomed to a blood pressure of 160 systolic, 100 diastolic, and when they were forced to work with a blood pressure of 80 systolic, 60 diastolic, they failed.

A PHYSICIAN: How about the epigastric pain?

DR. MALLORY: I have no explanation for that unless it was due to the spleen, which seems a little unlikely, or to the enormously enlarged retroperitoneal nodes around the celiac axis.

A PHYSICIAN: You did not examine the spine?

DR. MALLORY: We took sections from several vertebrae. We did not examine the cord. It is possible that she had a nerve-root involvement.

The New England
Journal of Medicine

Formerly the
Boston Medical and Surgical Journal
Established in 1828

OWNED BY THE MASSACHUSETTS MEDICAL SOCIETY AND PUBLISHED UNDER THE JURISDICTION OF THE COMMITTEE ON PUBLICATIONS

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SUBSCRIPTION TERMS: $6.00 per year in advance, postage paid, for the United States; Canada, $7.04 per year; $8.52 per year for all foreign countries belonging to the Postal Union.

MATERIAL for early publication should be received not later than noon on Saturday.

THE JOURNAL does not hold itself responsible for statements made by any contributor.

COMMUNICATIONS should be addressed to the NEW ENGLAND JOURNAL OF MEDICINE, 8 Fenway, Boston, Mass.

PROPOSED ADMINISTRATIVE ECONOMIES IN BOSTON

MAYOR TOBIN is to be congratulated on his stand in relation to economy in city administration. As it affects the medical and surgical care of the sick, his first step is, in our opinion, an excellent and long overdue one. The closing of the two relief stations that have for years encumbered the Boston City Hospital is, from the point of view of the individual patient's welfare, a most important and beneficial action. The equipment and personnel at both relief stations has never compared in adequacy with that available at the main hospital and, in the very nature of things, never could. On the other hand, and equally in the very nature of things, this personnel with its inadequate equipment was asked every day and night to face the most difficult and serious types of medical and surgical emergencies — emergencies that, when they did reach the main hospital, taxed to the utmost that staff with its specialists and modern equipment.

It has been argued that these stations should be kept open. Why? Because, say the proponents, the greater delay necessary to move patients to the main hospital will endanger their lives and welfare. There *was* a significant delay in the horse-drawn ambulance days, but with the modern police or hospital motor ambulance it no longer exists. Is there any other reason why the seriously injured citizens of Boston still must be forced to undergo the most important part of their treatment after a major injury, at the hands of an inadequate staff which is further handicapped by a shortage of equipment? Yes. Because these relief stations have served — particularly the East Boston one — as outpatient departments. If they are closed the local residents who need outpatient care will have to go to local physicians or to the Outpatient Department of the main hospital. This is a bother but is no more than that, and does not justify the expenditure of large sums by the city to save patients carfare, nor does it justify subjecting these same patients to an appreciably greater liability to death if and when they are struck by an automobile or fall into the hold of a ship.

This initial move, while most praiseworthy, must not be allowed to justify less meritorious economies. It has been reported in the daily press that the Boston City Hospital budget must be cut $400,000 in addition. It is hard to see how this can be done without curtailing or even eliminating services that the public has every right to expect and demand. Because these services have not the same news value as that of relief stations does not mean that they are less essential.

The trustees and the Mayor should recognize the futility and danger of forcing the organization at the Boston City Hospital to regress to the conditions of fifteen or twenty years ago. The trustees should not be permitted to do this, nor should the Mayor be allowed, through lack of understanding, to place himself in the position of demanding such a sacrifice even if no money is saved from the hospital budget.

STATE POSTGRADUATE COURSES

THE plan of the Committee on Postgraduate Education for the present year deserves commendation. Not only is a wide variety of subjects presented to the districts, but there is special emphasis on syphilis and gonorrhea, in collaboration with the program of the United States Public Health Service. "Early Syphilis" and "Gonorrhea in the Male" have been selected for presentation for the present year, with the intent of continuing in future years and eventually covering these subjects thoroughly. It is worthy of note that the course in syphilis has been dedicated to the late Dr. C. Morton Smith, whose interest and advice in the course before his death aided greatly in its preparation.

These courses are illustrated with many lantern slides, including charts and colored slides which show various phases of these diseases. They cover quite thoroughly the practical matters of diagnosis and treatment, and deserve a large attendance by practicing physicians in the various districts. Massachusetts physicians are now given an opportunity to become up-to-date on subjects about which more patients can be expected to ask questions in view of the publicity now being given to these diseases.

MASSACHUSETTS MEDICAL SOCIETY

SECTION OF OBSTETRICS AND GYNECOLOGY

M. FLETCHER EADES, M.D., *Secretary*
19 Bay State Road
Boston

CASE HISTORY NO. 58. BLEEDING AT TERM — PARTIAL SEPARATION OF THE PLACENTA

Mrs. G. R., a twenty-three-year-old primipara, was admitted to the hospital on October 1, 1937. She was at term, the membranes had ruptured one hour before admission, and she had experienced two or three attacks of mild abdominal pains, and had a slight bloody show.

The family history was negative. The patient's health had always been good. She had had measles as a child. The onset of menstruation occurred at thirteen. A regular thirty-day cycle was established, and the periods lasted three or four days without pain. The last menstrual period began on December 29, 1936.

The antenatal course was entirely normal. Her blood pressure remained at a level averaging 105 systolic, 70 diastolic. Repeated urinalyses were normal; the blood Wassermann was negative.

She was seen at 5 a. m. on the day of admission. Examination revealed a robust young woman in no apparent distress, with temperature 98.6°F., pulse 80 and respirations 22. The mucous membranes were of normal color. The thyroid gland was slightly enlarged. The lungs were normal to percussion and auscultation. The heart was normal in size; the sounds were regular and clear; no murmurs were heard. Abdominal examination revealed a pregnancy at term. The baby was large and presented as a right occiput anterior. The head was unengaged and could be moved above the pelvic inlet. The fetal heart sounds were normal. Rectal examination revealed a long, thick cervix, which admitted the tip of the examining finger. During the examination a moderate amount of blood-tinged amniotic fluid exuded. Examination of the blood showed a red-cell count of 3,950,000, a white-cell count of 7800 and a hemoglobin of 80 per cent (Sahli); the smear was normal.

Three hours after admission the labor pains were still mild and irregular. The blood loss was definitely in excess of the normal show. The head remained unengaged, and the fetal heart sounds normal. The patient was seen in consultation, and a diagnosis of partial premature separation of the placenta was made. A vaginal examination ruled out a placenta previa. As recommended by the consultant, the cervix and vagina were packed with iodoform gauze, an in-dwelling catheter was placed in the bladder, and a Spanish windlass was applied. The patient's condition at 11 a. m. was satisfactory; her pulse was strong, and the rate was 96. The blood pressure was 100 systolic, 60 diastolic.

In order to control the restlessness 1/6 gr. morphine sulfate and 9 gr. sodium amytal were given during the early afternoon. At 4 p. m. the patient was not definitely in labor, and 0.07 cc. (1 minim) obstetrical Pituitrin was ordered every half hour for three doses; active labor promptly followed. At 7 p. m. the patient began to complain of pressure on the rectum. The packing was removed, and no bleeding followed. Rectal examination revealed a moderately thick but soft cervix, which was three fingers dilated. The presentation was right occiput transverse, and the vertex was at the level of the ischial spines. During the evening

A series of selected case histories by members of the section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the section.

she was given 6 gr. sodium amytal. Throughout this time there was no vaginal bleeding, and the fetal heart sounds remained normal. At midnight the head was on the perineum. The voluntary effort in the second stage was poor; this was probably due to the medication. Solid blade forceps were applied to the head on the perineum; a median episiotomy was made, and a 9-lb. baby boy easily delivered. The infant cried spontaneously. The blood loss during the delivery did not exceed 300 cc., and the total blood loss was approximately 450 cc. The placenta was rectangular in shape. There was an adherent dark-red blood clot covering one angle.

The puerperium was afebrile, and the patient was discharged home on the twelfth postpartum day. The episiotomy was well healed, and the uterus had involuted normally. The baby had regained its birth weight.

Comment. Conservative treatment is of value in cases of premature separation of the placenta if the baby is dead, or very premature, if the mother is very toxic and the chances for a living baby are poor, if the mother is a poor operative risk or if labor has started and examination reveals a soft cervix with some dilatation, and if it is felt that the so-called Couvelaire uterus is not present. The conservative treatment consists of methods to control the bleeding, to improve the patient's general condition and to deliver the baby. The patient should be treated for shock, if it is present. The cervix and vagina should be packed tightly with iodoform gauze or sterile gauze, or a Voorhees's bag should be inserted, and a firm binder with a Spanish windlass should be applied to the abdomen. This usually controls the bleeding. Blood transfusion may then be resorted to, if indicated, or intravenous glucose may be given. Small doses of analgesic drugs should be administered, and when the cervix is fully dilated, the baby should be delivered. This case illustrates the use of conservative treatment.

However, if we have a living baby at term, in a primipara who has a long rigid, undilated cervix, delivery is best accomplished by an abdominal cesarian section. This also applies to patients in whom uteroplacental apoplexy (Couvelaire uterus) is suspected. In some cases of uterine apoplexy it may be necessary to resort to the Porro operation to control the bleeding.

MEDICAL POSTGRADUATE EXTENSION COURSES

The following sessions, given by the Massachusetts Medical Society in co-operation with the Massachusetts Department of Public Health, the United States Public Health Service and the Federal Children's Bureau, have been arranged for the week beginning February 14:

BRISTOL NORTH

Thursday, February 17, at 4:00 p. m., at the Morton Hospital, Taunton. Subject: The Use and Misuse of Prontylin. Instructor: Benjamin W. Carey, Jr. Arthur R. Crandell, *Chairman.*

BRISTOL SOUTH (New Bedford Section)

Friday, February 18, at 4:00 p. m., at St. Luke's Hospital, New Bedford. Subject: The Use and Misuse of Prontylin. Instructor: Benjamin W. Carey, Jr. Robert H. Goodwin and Howard P. Sawyer, *Chairmen.*

ESSEX SOUTH

Tuesday, February 15, at 4:00 p. m., in the Nurses' Home, Salem Hospital, Salem. Subject: Pneumococcus Pneumonia and Serum Therapy. Instructor: Maxwell Finland. Walter G. Phippen, *Chairman.*

MIDDLESEX EAST

Tuesday, February 15, at 4:00 p. m., at the Melrose Hospital, Melrose. Subject: Cesarean Section, Analgesia. Instructor: M. Fletcher Eades. Joseph H. Fay, *Chairman.*

MIDDLESEX NORTH

Friday, February 18, at 7:00 p. m., at St. John's Hospital, Lowell. Subject: Early Syphilis. Instructor: C. Guy Lane. William S. Lawler, *Chairman.*

NORFOLK

Friday, February 18, at 8:30 p. m., at the Norwood Hospital, Norwood. Subject: Atelectasis in the Newborn. Instructor: Clement A. Smith. Hugo B. C. Riemer, *Chairman.*

NORFOLK SOUTH

Monday, February 14, at 8:30 p. m., at the Quincy City Hospital, Quincy. Subject: Differential Diagnosis and Treatment of Scarlet Fever. Instructor: Edwin H. Place. David L. Belding, *Chairman.*

PLYMOUTH

Tuesday, February 15, at 4:00 p. m., in the Rosa Field Nurses' Residence, Brockton Hospital (rear of hospital), Brockton. Subject: Rheumatic Infection; Rheumatic Heart Disease. Instructor: Edward F. Bland. Walter H. Pulsifer, *Chairman.*

WORCESTER (Milford Section)

Thursday, February 17, at 8:30 p. m., in the Nurses' Home of the Milford Hospital, Milford. Subject: Toxemias of Pregnancy. Instructor: John Rock. Joseph Ashkins, *Chairman.*

WORCESTER NORTH

Friday, February 18, at 4:30 p. m., at the Burbank Hospital, Fitchburg. Subject: Differential Diagnosis and Treatment of Scarlet Fever. Instructor: Conrad Wesselhoeft. Edward A. Adams, *Chairman.*

CHILD BEHAVIOR

Since the behavior of children has been a matter of interest to adults from time immemorial, any attempt to deal with what we think we know about the subject must of necessity be very sketchy. Only a few of the high lights, especially those which have been cast by the careful observations of the past few years, can be mentioned.

Children's behavior depends so much upon their physical and mental development that we must consider in our discussion both these factors. Gessell at Yale has pointed out that there are types of activity which usually make up a part of the lives of all normal infants and children at about the same age. Thus the ability to roll over is the foundation stone upon which sitting up is later built, and the ability to understand pictures, peculiar to man and perhaps to the chimpanzee, is the first step toward the understanding and recognition of symbols which make reading possible. There is a growing suspicion that these newly appearing activities are the result of development of parts of the nervous system which are not mature at birth. Thus the normal new-born baby does not have to be taught to breathe or suck; if left to itself the need of air will cause it to breathe, and hunger when it comes will cause the baby to suck and swallow when suitable food is properly presented. We call such activities instinctive, but that merely means that the nervous machinery necessary for their carrying out is already present and "ready to go" at the time of birth. Indeed, as in most of nature's provisions, such leeway is provided that these fundamental activities take place effectively even though birth occurs as much as two months or more before term.

Certain activities which develop after birth are almost as automatic in the normal child as breathing and sucking. Among these may be mentioned sitting, standing and walking. Ordinarily they are not learned in the sense that reading is learned; the growing infant acquires them when he is ready, without teaching or encouragement. Those of us who feel that we must control the world for our children are tempted to try to vary the automatic schedule either by teaching the baby walking before he is ready (to keep him up with the Jones's baby) or by keeping him from walking when he is ready (to prevent his developing bowlegs). Walking develops at about its own pace in either case, and attempts to interfere with it often disturb the baby's disposition.

Many of the other acquired activities of infancy and childhood are largely automatic and the result of ripening processes in the nervous system. If not interfered with by ambitious or solicitous parents, they appear in an orderly fashion, but if attempts are made to force them, resistances and emotion are often built up which delay or modify their appearance. It is impossible to describe all these activities but as examples may be mentioned such things as being weaned from the bottle to the cup, chewing and swallowing solid foods, and the like. Of course, if the baby is never given the cup or solid foods, he will not learn to deal with them, but if too much interest and effort are centered on teaching their use too early, resistance to them is often established. Such an attitude of resistance may delay the accomplishment of the desired activity for a long time, and may influence the personality of the child toward his family or the world in later life.

The control of bowel movements and urine is a matter over which many parents fuss too much. All dog trainers know that it is useless to try to housebreak a puppy before it has attained an age of several months, but parents are often unwilling to wait a reasonable time before attempting so to train their children. Everyone knows that people accustomed to handling babies can prevent their soiling themselves in the day by having them use a chamber from the time they are a few months old, but this is not so much training the baby as it is getting the nurse to learn when a movement may be expected and acting accordingly. It is also possible to keep a toddler dry in the day by having an adult handy to put him on the toilet every fifteen minutes or half hour or however often he needs to go, an interval usually ascertainable by observation. This is not education so far as the baby is concerned and may well rouse antagonism on the baby's part to an extent making it wise to abandon attempts to teach toilet habits at this age. Even after the child has learned to ask to go, it is important to realize that the interval between his warning sensation and the need for relief is so short that it may be impossible for him, in spite of the best intentions, to reach the toilet in time.

Bed-wetting is a type of infantile behavior often carried over into childhood, often because of too early enthusiastic efforts to stop it. Dry nights may become habitual as a result of picking up two-year-old children at adult bed time and taking them to the toilet. Many children do not, however, respond to this treatment, and it is most important that their wet beds shall not be considered a matter of interest until they are three years old or more. Realization of these facts would, of course, not prevent all the wet beds in Christendom, but would certainly reduce their number.

The older children become, the more important teaching is in their learning new activities. In spite of this, the fact that "growing up" is still going on in the brain must never be forgotten. Reading is such an activity. No one would expect most three- or four-year-old children to learn to read; some children are ready to learn at four or five, most by six and an appreciable number, who are otherwise quite normal, not until they are eight or nine. An occasional child will pick up reading from magazines about the house or from his brothers and sisters at home, but for most children reading is an acquired art learned only through the medium of suitable material properly presented. The point I wish to make is that until the child is ready to learn to read, it makes no difference how skillfully the subject is presented to the child; he cannot make use of it. Psychologists and schoolteachers have recently been working out tests to show whether a child is ready for reading. If these tests could be applied to all children before any attempt to teach reading is made, and if parents could be persuaded to accept the results placidly, it would cut out many, many hours of heartache and the resultant behavior difficulties.

It is therefore of the utmost importance for parents to realize that their place in the care of their infants and children is in supplying to them food, clothing, shelter and affection. The fact that it is so much more convenient to have food prepared at stated times and that the busy mother or nurse can bathe and dress the child at only certain times, will automatically teach him a little about getting on with the world. Little children will learn most of what they need to learn either automatically or by imitation. If at appropriate ages they have not, then careful investigation of why they do not is in order.

* * *

Q. I have a family of several children about whom I should like to ask you. In the first place my baby of twelve months is not yet walking. When I see my neighbor's baby, two months younger, walking, I wonder

A "Green Lights to Health" broadcast given by Dr. Randolph K. Byers on Wednesday, January 26, and sponsored by the Public Education Committee of the Massachusetts Medical Society and the Massachusetts Department of Public Health.

whether my baby has spinal trouble or whether his brain is all right. What should I do about it?

A. There is a great difference in the age at which normal babies learn to walk. If the baby seems otherwise active and normally interested in his surroundings, have patience. If you think he is not so active as he should be, take him to your doctor for advice, but do not try to urge him to walk, without advice.

Q. Washing and sterilizing bottles is so much trouble that I have taught this baby to drink from a cup. Lately I notice he is sucking his thumb a lot. What should I do about it?

A. Sucking is to a large extent an important emotional satisfaction to infants, the need for which is outgrown at variable ages. Your baby probably still needs it. If you let him get his satisfaction in the way he was meant to in getting his food, he may be less tempted to get it by sucking his thumb. In any case, thumb-sucking at this age is so common that strenuous attempts to break the habit are unnecessary.

Q. My boy of four is so lazy he will not learn to dress himself. How can I teach him?

A. Many children of four are not yet sufficiently skillful with their fingers to fasten buttons. Get him the sort of underwear held on by soft elastic strips around the shoulders and let him put that on while you fasten the buttons of his outer clothing.

Q. My two-year-old is terrible, whatever I ask of him he says, "No."

A. One of the striking characteristics of many two-year-olds is so-called negativism. Try to warn him that you are going to make a request a few minutes before you make it, and then make it in a quietly confident manner. Be very sure to make just as few requests as possible.

Q. I have tried to train this child to move his bowels every day at the same time, but sometimes he moves them twice one day and not at all the next. How can I train him?

A. Regularity of bowel movement at his age is not essential, though very desirable. If his stools do not become hard, let him move his bowels when he feels the need. Very often the price paid in emotional upset by the baby and parent in securing absolute regularity of bowel movement is not worth the result.

* * *

In summary I want to stress the fact that the development of new activities in babies and young children comes not as the result of teaching but as the result of development and growth in the brain and nerves attached to it. Attempts to interfere with the unfolding of the normal schedule of activity, if carried on strenuously, tend to rouse resistances and dislikes in babies, which very often affect their personalities in childhood and adult life. The temptation to send children to school before their brains have matured to the point which allows them to understand schoolwork is very apt to have the same sort of effect. Mistakes along these lines on the part of parents are very apt to cause unsatisfactory behavior in their children, and tolerance on the part of parents for slow development in the babies is often rewarded by children with stable dispositions.

MISCELLANY

DR. VAUGHAN TO ADDRESS MEETING OF BOSTON TUBERCULOSIS ASSOCIATION

Dr. Henry F. Vaughan, who has been the health commissioner of Detroit, Michigan, since 1919, will be the speaker at the annual meeting of the Boston Tuberculosis Association which will be held at the Hotel Copley-Plaza on February 15, at 4 p. m. Dr. Vaughan has been with the Detroit Health Department since 1914, first serving as sanitary engineer and later as assistant health commissioner. During the World War he dropped out of the department to serve as captain in the Sanitary Corps, United States Army. At Camp Upton he was the camp epidemiologist, then, together with Major Russell L. Cecil, he was commissioned by Surgeon General Gorgas to make a special study of the epidemiology and prevention of pneumonia at Camp Wheeler. Dr. Vaughan is a trustee of the W. K. Kellogg Foundation with headquarters in Battle Creek, Michigan. This foundation, chartered to improve the health, welfare and happiness of children, is conducting a community-health-service program in seven rural counties of southwestern Michigan. An effort is being made to co-ordinate all the resources of these communities for the improvement of child health. The foundation is spending approximately $1,000,000 annually in this program. Dr. Vaughan has been particularly interested in recent years in advancing the so-called plan of medical participation — the purpose of which is to extend the influence of the community-health service by integrating therein the facilities and services of the physician and dentist in private practice. This type of program is proposed as an adjunct to the fundamental services of a whole-time local health department. Dr. Vaughan has served on innumerable committees, both administrative and research, of the American Public Health Association, of which organization he was president in 1925. He has also served on the boards of many other national health organizations. He has been a teacher of public health administration since 1915 and is now professor of public health administration at Wayne University, Detroit, and a special lecturer at Vanderbilt University and the University of Michigan.

RÉSUMÉ OF COMMUNICABLE DISEASES IN MASSACHUSETTS FOR DECEMBER, 1937

DISEASES	DECEMBER 1937	DECEMBER 1936	FIVE-YEAR AVERAGE*
Anterior poliomyelitis	1	0	6
Chickenpox	1671	1393	1488
Diphtheria	18	18	78
Dog bite	574	497	413
German measles	72	63	108
Gonorrhea	495	590	579
Lobar pneumonia	411	557	504
Measles	349	2006	1182
Meningococcus meningitis	4	12	8
Mumps	298	663	608
Paratyphoid A	0	0	0
Paratyphoid B	3	1	0
Scarlet fever	928	753	956
Syphilis	475	444	401
Tuberculosis, pulmonary	341	203	261
Tuberculosis, other forms	43	25	26
Typhoid fever	19	5	10
Undulant fever	8	3	2
Whooping cough	732	1738	899

*Based on figures for preceding five years.

RARE DISEASES

Anterior poliomyelitis was reported from Chicopee, 1.

Diphtheria was reported from: Athol, 1; Boston, 1; Brockton, 2; Cambridge, 1; Chelmsford, 2; Chelsea, 1; Fall River, 3; Framingham, 1; Lawrence, 1; Lynn, 1;

Peabody, 1; Southbridge, 2; Tewksbury, 1; total, 18.

Dysentery, amebic, was reported from Newton, 1.

Dysentery, bacillary, was reported from Boston, 1; Cambridge, 9; Fall River, 4; New Bedford, 3; Revere, 1; Wellesley, 1; total, 19.

Encephalitis lethargica was reported from Northampton, 1.

Leprosy was reported from New Bedford, 1.

Meningococcus meningitis was reported from Melrose, 1; Pittsfield, 1; Rockland, 1; Tyngsboro, 1; total, 4.

Paratyphoid B was reported from Boston, 1; Greenfield, 1; Monterey, 1; Revere, 1; total, 4.

Pfeiffer bacillus meningitis was reported from Westfield, 1.

Septic sore throat was reported from Beverly, 1; Boston, 2; Cambridge, 2; Gardner, 4; Lawrence, 1; Rutland, 1; Somerville, 1; Wellesley, 1; total, 13.

Tetanus was reported from Amherst, 1.

Trachoma was reported from Everett, 1; Plymouth, 1; total, 2.

Trichinosis was reported from Springfield, 1.

Typhoid fever was reported from Belmont, 1; Cambridge, 1; Lowell, 1; Medford, 1; Sharon, 1; Somerville, 1; Stoughton, 13; total, 19.

Undulant fever was reported from Boxboro, 1; Brookline, 1; Fitchburg, 1; North Adams, 1; Sterling, 1; Taunton, 2; Westfield, 1; total 8.

Diphtheria continued to show low incidence.

Measles, mumps, German measles, and scarlet fever were reported below the five-year average.

Paratyphoid fever continued to show record high incidence.

The reported incidences of anterior poliomyelitis, meningococcus meningitis, lobar pneumonia and whooping cough were below the five-year average.

Undulant fever showed record high incidence.

Pulmonary tuberculosis, chickenpox and tuberculosis (other forms) were reported above the five-year average.

The increase in the reported incidence of typhoid fever was associated with a recent outbreak in Stoughton; epidemiological investigation implicated a banquet, the food of which had been prepared by a carrier.

Animal rabies showed record low incidence. New foci were reported in Fitchburg and Westborough.

CAMBRIDGE MUNICIPAL TUBERCULOSIS HOSPITAL

Plans have been announced whereby the present Cambridge Home for the Aged and Infirm will be converted into a tuberculosis hospital. In order to meet the minimum standards of the Massachusetts Department of Public Health it was estimated that an outlay of nearly $400,000 would have been required to build and equip an additional wing to the present sanatorium. The plan has been approved by the Commissioner of Public Health, and the former inmates of the home will be cared for in other institutions, nursing homes and private homes.

CONGRESS ON MEDICAL EDUCATION AND LICENSURE

The thirty-fourth annual Congress on Medical Education and Licensure will be held at the Palmer House, Chicago, on February 14 and 15. The first day will be devoted to papers and discussions on medical education; Dr. Ray L. Wilbur, of Stanford University, will preside at the morning session, and Dr. Frederic A. Washburn, of Boston, in the afternoon. Of the two morning sessions on the second day, one covering postgraduate education will be in charge of Dr. Charles G. Heyd, of New York City, while Dr. Jesse W. Bowers, of Fort Wayne, Indiana, will preside at a joint meeting of the Council on Medical Education and Hospitals and the Federation of State Medical Boards. The afternoon session of the Federation of State Medical Boards will be a symposium on promulgation of regulations authorized by law, and will be in charge of Dr. Bowers.

REVISION OF PHARMACOPOEIA TESTS

The following revision of the U.S.P. XI tests is hereby announced through the authority of the Committee of Revision and the action of the Board of Trustees.

Aqua Aurantii Florum. Page 64.

Change the test for residue on evaporation to: "Evaporate 100 cc. of the Orange Flower Water on a water bath, and dry the residue to constant weight at 100°C.: not more than 0.005 gm. of residue remains."

Benzoinum. Page 86.

Omit the test for "rosin," lines 13 and 14 from the bottom of the page.

Oleum Amygdalae Expressum. Page 248.

Omit the test for the solidification point of the fatty acids, lines 9 and 10 from the bottom of the page.

The urgency of the revision of these three tests, due to their involving the importation of these official items, necessitates an interim revision announcement. It is expected that from time to time other revisions in official texts will be similarly announced, to meet conditions which demand prompt action.

When enough of these interim revision announcements have accumulated and other added or revised monographs have been adopted it is expected that all these new and revised texts, approved since the appearance of the *First U.S.P. XI Supplement,* will be reprinted in full and issued as the *Second U.S.P. XI Supplement.* Interim revisions are being announced through the medical and pharmaceutical press.

E. Fullerton Cook, *Chairman,*
Committee of Revision
of the *U. S. Pharmacopoeia.*

MEDICAL ART ASSOCIATION

The American Physicians' Art Association, a national organization of medical men who have ability in the fine arts, will hold a first national exhibition in the San Francisco Museum of Art, San Francisco, California, in June, 1938. (The American Medical Association Convention is June 13-17 in the same city.) The American Physicians' Art Association already has an outstanding membership. There are three classifications for membership: active, associate and contributing. The first annual exhibition promises to be of unusual interest with entries to be accepted (after jury selection) in the following classifications: oils, watercolors, sculpture, photography, pastels, etchings, crayon and pen and ink drawings (including cartoons), wood carvings and book bindings. Scientific medical art work will not be accepted. The exhibition is not limited to first showings. All entries close April 1, 1938. Any physician interested should communicate at once with the Secretary of the American Physicians' Art Association, Suite 521-536 Flood Building, San Francisco, California.

CORRESPONDENCE

MEDICAL RECORDS

To the Editor: The question as to what provision a physician or surgeon should make in regard to his medical records is one which I should like to have help in solving. I am writing this letter in hope that some of the readers of the *Journal* will have sufficiently concrete ideas so that a policy may be formed.

The whole subject was recently brought forcibly to my attention when I saw a doctor's heirs offering for sale his files with the records in them. This was in a small town where several prospective purchasers would have been glad to obtain news about the neighbors. Fortunately, this catastrophe was averted, but it made me think how I should protect my own records.

With the growth of scientific medicine and the careful investigations that many doctors give their patients, combined with the detailed histories, x-ray findings, laboratory reports and records of operations, I believe that a certain proportion of our records would be of real value to the doctor who treats these patients from where we leave off. Would it be feasible for a doctor to leave his records to a small group of physicians with a sufficient sum of money to pay for their storage with the understanding that that group would look through a record before giving it out to the physician to whom it might be of help so that personal letters and irrelevant statements could be first deleted? I have personally had patients referred to me, whose previous surgeon had died, where the pathological findings and procedures of a previous operation would have been very helpful. I believe that in straight medical cases the past histories and laboratory findings would be equally useful to a physician who was carrying on.

At present, I can see no course open to us as physicians but the simple one of burdening our nonmedical families with the responsibility of our records and the request that they be destroyed at the end of a certain period of time.

HILBERT F. DAY, M.D.

412 Beacon Street,
Boston.

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

To the Editor: In addition to the articles enumerated in our letter of December 4, the following have been accepted:

Mallinckrodt Chemical Works
Hippuran, 100 gm. bottle
Hippuran, 500 gm. bottle

Sharp & Dohme
Rabies Vaccine (Phenol Killed) (Semple) 14 dose package, with syringe

John Wyeth & Brother, Inc.
Vaginal Suppositories Silver Picrate — Wyeth's, 1 gr. (infant size)

PAUL NICHOLAS LEECH, *Secretary.*

535 North Dearborn Street,
Chicago, Illinois.

MEDICAL SERVICE IN CHINA

To the Editor: The Emergency Medical Committee for China of the Foreign Missions Conference of North America has been set up for the purpose of finding a group of selected young surgeons for service in China to relieve the terrific pressure put on existing medical institutions in caring for war wounded. The need for immediate assistance can hardly be exaggerated, as many thousands of soldiers and civilians are undergoing untold suffering, being still uncared for in every way.

I am taking the liberty to ask your assistance in canvassing for available young men with surgical experience — married or single — who could go out alone for at least one year for such service as above indicated.

This committee is offering expenses paid to and from the Far East, and a salary equal to the average now being paid to medical workers in China.

We shall especially appreciate the recommendation of individuals who you think would be suitable and interested in humanitarian service, and I shall be glad to arrange for personal interviews, either here or elsewhere, with any candidates that you might recommend.

S. H. LILJESTRAND, M.D.,
Executive Secretary.

Room 811, 150 Fifth Avenue,
New York City.

RECENT DEATHS

LARSON — CARL G. LARSON, M.D., of 119 High Street, Medford, died December 23. He was in his forty-third year.

Dr. Larson received his degree from the College of Physicians and Surgeons in Boston in 1927.

He was a fellow of the Massachusetts Medical Society and a member of the American Medical Association.

LEARD — JOHN S. LEARD, M.D., of West Roxbury, died at his home, February 3, after a week's illness. He was in his seventy-second year.

A native of Prince Edward Island, he graduated from Prince of Wales College and received his degree from the University of Pennsylvania School of Medicine in 1894.

Dr. Leard was a member of the original staff of the Faulkner Hospital and was organizer and first president of the West Roxbury-Roslindale-Jamaica Plain Medical Association. He was a fellow of the Massachusetts Medical Society and the American Medical Association. At the time of his death he was vice-president of the Jamaica Plain Dispensary and a member of the Clinical Club of Boston and of the Norfolk District Medical Society of which he was a past president, councilor and censor.

Among his other affiliations were memberships in the Masonic Order, the Unitarian Club of West Roxbury, the Emmanuel Men's Club and the Highland Club of West Roxbury.

His widow, a daughter and twin sons survive him.

REPORT OF MEETING

NEW ENGLAND HEART ASSOCIATION

The regular monthly meeting of the New England Heart Association was held at the Peter Bent Brigham Hospital on January 24. The following program was presented:

THE DEVELOPMENT OF FATAL LUETIC AORTITIS WITHIN TWO YEARS AFTER PRIMARY INFECTION. Maurice A. Schnitker, M.D.

The case was that of a Canadian-born housewife, age twenty-three, who, except for two operations, was cared for in a distant hospital. Married at sixteen, she went through normal pregnancies at seventeen and at eighteen. In the next three years she was hospitalized for breast abscess, acute gonococcal salpingitis and minor illness, during which time three blood tests for syphilis were negative. Examination of the heart during this period was entirely normal. At the age of twenty-one, one and a half years after the gonococcal infection, she contracted syphilis, being seen in the secondary stage of the disease, with Wassermann, Kahn and Hinton tests positive. She was treated for the next seven months, irregularly, with sulfarsphenamine; this was essentially the only treatment received. Six months after the onset of the syphilis, while under treatment, she began to have typical angina pectoris with radiation of pain to both arms and hands. At that time the heart was enlarged, and revealed coarse, systolic and diastolic murmurs of aortic insufficiency and an Austin-Flint sound. Nitroglycerin gave some relief. At twenty-two, the pains were incapacitating, and she entered the Peter Bent Brigham Hospital where left cervicothoracic sympathectomy was done with temporary complete relief of pain. At this time physical examination and x-ray showed, in addition, a large aneurysm of the aorta. Electrocardiograms revealed only left-axis deviation. Two months later, with severe recurrence of pain in the right shoulder, arm and back, she re-entered the hospital and a right sympathectomy was performed. Pain recurred several weeks later, with severe dyspnea and hemoptysis. She died in an attack of pain at the age of twenty-three, two years after the onset of syphilis and eighteen months after the beginning of cardiac symptoms. At necropsy, the heart weighed 790 gm. and showed a huge aneurysm of the aorta, syphilitic aorta insufficiency and practically complete occlusion of the right coronary ostium.

Sources of Error in the Direct Method of Venous Pressure. Richard H. Lyons, M.D., J. Allen Kennedy, M.D., and C. Sidney Burwell, M.D. Presented by Dr. Lyons.

There is wide variation in the reports of venous-pressure measurements in normal people. This results from a lack of uniformity of methods used and from variations in the position of the reference point. In an effort to define more closely the normal limits of venous pressure, certain sources of error in the direct method have been considered. The position of the site of venipuncture, the bore of the manometer and variations in the physiologic state of the subject may distort the venous-pressure readings. Venous pressures were determined on 90 normal subjects, and the suitability of the reference points of von Recklinghausen, Moritz and Tabora, and Eyster. It was found that the use of reference points involving the thoracic diameter of the subject gave lower venous-pressure readings in subjects with larger chests. This may be eliminated by using the table top as the reference point with the patient in the supine position.

Physiologic Effects of Artificial Fever. John G. Gibson, 2nd, M.D.

The physiologic effects of artificial fever can be interpreted in light of the great increase over normal in heat exchange. Extreme vasodilatation occurs in the effort to maintain normal temperature, with increase in pulse rate, blood flow, venous pressure and cardiac output, resulting in an increased volume capacity.

Sweating is profuse, and plasma volume reduced, the degree of reduction being related to the amount of fluids given. Since fluid absorption from the gastrointestinal tract may be retarded, there is a tendency toward a disparity between the volume capacity and circulating volume.

Gross water loss is great, and is determined by the temperature, humidity and air movement of the patient's environment, being least in cabinets in which humidity is highest, dry-bulb temperature lowest and air velocity least. Tissue-fluid loss is determined by the rate of fluid intake, and a critical level exists for each patient beyond which further loss involves risk of peripheral vascular collapse.

The loss of chlorides in sweat and of carbon dioxide through hyperventilation brings about an alkalosis, the degree of which is related to the degree of dehydration permitted to occur.

Provided water, chlorides and carbon dioxide are administered in amounts adequate for replacement of losses, high temperatures (106.7°F.) may be safely maintained for long periods (6 to 15 hr.).

The Antagonism of Hypertension and Active Tuberculosis. A. W. Contratto, M.D.

In approximately 32,000 individuals admitted to the Medical Service of the Peter Bent Brigham Hospital, from 1913 to 1936 inclusive, there were 1250 cases of pulmonary tuberculosis, 761 of which were active.

The average age of the entire group was 42.9 years, of the active male group 40.5 years, and of the active female group 35.5 years.

The average blood-pressure determinations in the various decades (second to eighth) were from 10 to 15 mm. lower in both the systolic and diastolic readings in the active tuberculous groups than the considered average pressures of normal persons. There were only 6 individuals, or 0.8 per cent, in the active group who had a systolic blood pressure of more than 160 mm. of mercury, and the majority of these cases were not diagnosed as tuberculosis clinically and even at autopsy there was some doubt as to the activity of the lesion.

There were 4 cases of rheumatic heart disease in the active group. Two of these came to autopsy. One had aortic insufficiency and no mitral stenosis, the other had mitral stenosis only. The other 2 cases were diagnosed as mitral stenosis clinically. In the active group there were no cases of angina pectoris or cardiac infarction.

Three hundred and thirty-two cases came to autopsy. One hundred and twenty-four of these were active cases of pulmonary tuberculosis. The average heart weight was much less in the active than in the healed cases, even in proportion to body weight.

It seems that all forms of cardiovascular disease exist only rarely in persons with active tuberculosis.

Observations on the Fourth Lead of the Electrocardiogram. Roger W. Robinson, M.D., A. W. Contratto, M.D., and S. A. Levine, M.D. Presented by Dr. Robinson.

Nine different chest leads were chosen covering the cardiac silhouette to determine if there was one position which would give reliable information in a great majority of instances and could be used as a standard fourth lead. Five hundred tracings were taken on 350 patients, of whom 76 came to autopsy. The old polarity was used. It was found that an upright T wave occurs frequently along the sternum but only rarely at the apex in normal

hearts. When an upright T wave occurs as the only abnormality in the fourth lead, it is not reliable evidence for myocardial infarction. Large hearts may show no Q wave in any precordial position, except the apex, without myocardial infarction. The Q wave may not disappear for fifteen days after an acute anterior infarction, and then it may disappear only at the apex. An absent Q wave at the apex is practically always associated with an anterior myocardial infarction. The fourth lead may be correctly interpreted in the presence of auricular fibrillation and delayed intraventricular conduction. We believe that the apex should be used as the point of application of the electrode for the standard fourth lead.

NOTICES

TUMOR CLINIC, BOSTON DISPENSARY

Each Tuesday and Friday morning from ten to twelve-thirty there is a meeting of the Tumor Clinic of the Boston Dispensary, a unit of the New England Medical Center. All kinds of tumors are seen, discussed, and when indicated, treated with radium and high-voltage x-ray.

Physicians are welcome to visit this clinic and bring a patient to the clinic for diagnosis.

AMERICAN BOARD OF OPHTHALMOLOGY

During 1938 the American Board of Ophthalmology will hold examinations as follows:

San Francisco, June 13,
Washington, D. C., October 8,
Oklahoma City, November 15.

Applications should be filed immediately. The required number of case reports must be filed at least sixty days prior to the date of examination. Application blanks can be procured from: Dr. John Green, 3720 Washington Avenue, St. Louis, Missouri.

FEVER THERAPY

The Peter Bent Brigham Hospital is now equipped to give fever therapy induced by physical means. This is under the immediate supervision of Dr. John G. Gibson, 2nd. Physicians desiring to send patients to the hospital for fever therapy should arrange by telephone with the admitting physician; he will furnish information as to charges for such service. Advice in regard to the use of fever therapy, suitability of individual patients for it, and so forth, can be obtained by telephoning Dr. Gibson at the Peter Bent Brigham Hospital; he should be communicated with before final arrangement for admission of the patient has been made.

POSTGRADUATE INSTITUTE OF THE PHILADELPHIA COUNTY MEDICAL SOCIETY

The Third Annual Postgraduate Institute, offering an intensive and interesting study of diseases of the digestive tract, will be conducted by the Philadelphia County Medical Society from March 28 to April 1 inclusive.

The program to be held in the Bellevue-Stratford Hotel, Philadelphia, has been designed to meet the needs of all members of the profession, but particularly those in general practice.

Physicians from fourteen states having attended last year's institute, an invitation to attend the 1938 session has been extended to the members of all county societies.

Lecturers have been selected from among the foremost teachers in this great medical center. While approaching the subject from specialized viewpoints, the presentations will be of a strictly practical nature and of real value to the general practitioner, who finds that digestive conditions occupy a considerable portion of his time.

The Philadelphia County Medical Society, in conducting the institute, is meeting the demands of many physicians who believe that the organized profession should provide them with this type of opportunity for keeping abreast of medical progress, and thus maintaining the highest standards of medical service.

The only charge is a $5.00 registration fee to cover the institute's expenses.

Additional information may be secured from your county society or from the Philadelphia County Medical Society, 21st and Spruce Streets, Philadelphia.

UNITED STATES CIVIL SERVICE EXAMINATION

Junior Scientific Aid (Parasitology), $1440 A Year

Applications must be on file with the United States Civil Service Commission at Washington, D. C., not later than February 28.

Applicants must show that they have had at least two years of experience in a zoological laboratory. The required experience must have been full time and paid. Applicants may substitute one year of study including a course in zoology, successfully completed in a college or university of recognized standing, for one year only of the required experience.

CAMBRIDGE HOSPITAL

The regular clinicopathological meeting of the staff of the Cambridge Hospital will be held at the hospital, 300 Mt. Auburn Street, Cambridge, on Tuesday, February 15, at 8:30 p. m.

All members of the medical profession are cordially invited to attend.

JOSEPH M. WADDEN, M.D., *Secretary.*

SOUTH END MEDICAL CLUB

The next regular meeting of the South End Medical Club will be held at the headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston, on Tuesday, February 15, at 12 noon.

Dr. William Dameshek will speak on "Hematological Problems in General Practice."

All physicians are cordially invited to attend.

JOHN B. HALL, M.D., *Secretary.*

NEW ENGLAND PATHOLOGICAL SOCIETY

A meeting of the New England Pathological Society will be held in the amphitheater of the Mallory Institute of Pathology, Boston City Hospital, on Thursday, February 17, at 8:00 p. m.

PROGRAM

Alzheimer's Disease. Dr. Naomi Raskin.

The Vascular Pattern in Various Lesions of the Human Central Nervous System: Studies with the benzidine stain. Dr. Leo Alexander, Dr. T. H. Suh and Dr. Tracy J. Putnam.

Time Determination of Histopathologic Changes in the Brain: Experimental incisions into the brain with transection of meningeal and intracerebral blood vessels in cats. Dr. Leo Alexander and Dr. Hugh P. Newbill.

Physicians and medical students are cordially invited to attend.

J. B. HAZARD, M.D., *Secretary.*

NEW ENGLAND SOCIETY OF PHYSICAL MEDICINE

The regular meeting of the New England Society of Physical Medicine will be held at the Hotel Kenmore, Boston, on Wednesday evening, February 16, at 8:00 o'clock.

Dr. Robert S. Harris will talk on "Medical Application of Radiant Energy." Discussion by Dr. Franklin P. Lowry.

The Council will meet at 6:00, and dinner will be served in the Empire Room at 6:30.

All members of the medical profession are cordially invited to attend.

WILLIAM D. MCFEE, M.D., *Secretary.*

BOSTON SOCIETY OF PSYCHIATRY AND NEUROLOGY

The next meeting of the Boston Society of Psychiatry and Neurology will be held at the Boston Medical Library, Thursday evening, February 17, at 8:15 o'clock.

PROGRAM

Chronic Progressive Chorea with Senile Changes in the Cortex and Basal Ganglia: Case report. Dr. David Rothschild.

The Effect of Benzedrine Sulfate on Sleep, Normal and Drug Induced. Dr. Abraham Myerson.

Experiences with Meningiomas of the Brain. Dr. Gilbert Horrax.

H. HOUSTON MERRITT, M.D., *Secretary.*

SOCIETY MEETINGS AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, FEBRUARY 14

TUESDAY, FEBRUARY 15

*9-10 a. m. Boston Dispensary. Clinicopathological conference. Dr. R. C. Wadsworth.

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

*12 m. South End Medical Club. Headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston.

4 p. m. Boston Tuberculosis Association. Hotel Copley-Plaza.

5 p. m. Cutter Lecture on Preventive Medicine. Harvard Medical School Amphitheater Building E.

WEDNESDAY, FEBRUARY 16

*9-10 a. m. Boston Dispensary. Hospital case presentation. Dr. S. J. Thannhauser.

*12 m. Clinicopathological conference. Children's Hospital Amphitheater.

*8 p. m. New England Society of Physical Medicine. Hotel Kenmore, Boston.

THURSDAY, FEBRUARY 17

8:30-9:30 a. m. Exchange visit, surgical and orthopedic staffs of the Peter Bent Brigham and Children's hospitals, held this week at the Children's Hospital.

*9-10 a. m. Boston Dispensary. Hyperventilation Syndromes. Dr. Maurice Sokolow.

*8 p. m. New England Pathological Society. Amphitheater of the Mallory Institute of Pathology, Boston City Hospital.

8:15 p. m. Boston Society of Psychiatry and Neurology. Boston Medical Library, 8 Fenway, Boston.

FRIDAY, FEBRUARY 18

*9-10 a. m. Boston Dispensary. Some Recent Developments in Purpura and Hemophilia. Dr. Frederick J. Pohle.

*10 a. m. - 12:30 p. m. Tumor clinic. Boston Dispensary.

12 m. Clinical meeting of the Children's Medical Service, Massachusetts General Hospital, Ether Dome.

SATURDAY, FEBRUARY 19

*9-10 a. m. Boston Dispensary. Hospital case presentation. Dr. S. J. Thannhauser.

*10 a. m. - 12 m. Staff rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

SUNDAY, FEBRUARY 20

4 p. m. Illustrated, public, health lecture, Faulkner Hospital auditorium. Progress in Dental Surgery. Dr. Kurt H. Thoma.

4 p. m. Free public lecture. Harvard Medical School, amphitheater of Building D. Overweight and Underweight. Dr. F. Dennette Adams.

4 p. m. Free public lecture. Beth Israel Hospital, Boston, in conjunction with the Women's Auxiliary. Dangers that Lurk in Cosmetics. Dr. Jacob J. Schwartz.

*Open to the medical profession.

FEBRUARY 14 and 15 — Congress on Medical Education and Licensure. Page 279.

FEBRUARY 14 — American Board of Internal Medicine. Page 969, issue of December 9.

FEBRUARY 15 — Cambridge Hospital. Clinicopathological meeting of the staff. Page 282

FEBRUARY 15 — Boston Tuberculosis Association. Page 244, issue of February 3, and page 278.

FEBRUARY 15 — Cutter Lecture on Preventive Medicine. Page 245, issue of February 3.

FEBRUARY 15 — South End Medical Club. Page 282.

FEBRUARY 16 — New England Society of Physical Medicine. Notice above.

FEBRUARY 17 — Boston Society of Psychiatry and Neurology. Notice above.

FEBRUARY 17 — New England Pathological Society. Page 282.

FEBRUARY 21 — Boston Medical History Club. 8:15 p. m., Boston Medical Library, 8 Fenway.

FEBRUARY 22 — New York University College of Medicine. Alumni Day. Page 245, issue of February 3.

FEBRUARY 25 — Massachusetts Psychiatric Society. Page 245, issue of February 3.

MARCH 1 — Greater Boston Medical Society. Beth Israel Hospital auditorium, 8:30 p. m.

MARCH 3 — George Washington Gay Lecture. Page 245, issue of February 3.

MARCH 10 — Pentucket Association of Physicians. Hotel Bartlett, 95 Main Street, Haverhill, 8:30 p. m

MARCH 10, 11, 12 — New England Hospital Association. Page 51, issue of January 6.

MARCH 28 - APRIL 1 — Postgraduate Institute of the Philadelphia County Medical Society. Page 282.

APRIL 4-8 — The American College of Physicians. Page 41, issue of July 1.

MAY 31, JUNE 1 and 2 — Annual meeting of the Massachusetts Medical Society. Hotel Bradford, Boston.

JUNE 13-17 — American Medical Association. San Francisco.

JUNE 13, OCTOBER 8 and NOVEMBER 15 — American Board of Ophthalmology. Page 282.

OCTOBER 17-21 — Clinical Congress of the American College of Surgeons, New York City.

DISTRICT MEDICAL SOCIETIES

BRISTOL SOUTH

MAY 5 — 5 p. m., New Bedford.

ESSEX SOUTH

MARCH 2 — Lynn Hospital. Clinic at 5 p. m. Dinner at 7 p. m. Speaker and subject to be announced.

APRIL 6 — Gloucester Hospital, Gloucester. Clinic at 5 p. m. Dinner at 7 p. m. Speaker and subject to be announced.

MAY 5 — Censors meet at Salem Hospital, 3:30 p. m.

MAY 11 — Annual meeting, Salem Country Club, Peabody. Dinner at 7 p. m. Speaker and subject to be announced.

FRANKLIN

Meetings will be held at the Franklin County Hospital, Greenfield, at 11 a. m. the second Tuesdays of March and May.

HAMPDEN

Meetings will be held on the fourth Tuesday in April and July.

MIDDLESEX EAST

Meetings will be held at the Bear Hill Golf Club, Stoneham, at 12:15 p. m. on March 16, and May 11.

MIDDLESEX NORTH

Meeting will be held at the Vesper Country Club, Lowell, on April 27.

NORFOLK DISTRICT

FEBRUARY 23 — Hotel Kenmore. 8:15 p. m. Dermatitis Venenata Due to Cosmetics and Industrial Irritants. Dr. John G. Downing. Discussion by Dr. Francis P. McCarthy.

MARCH 29 — Hotel Kenmore. 8:15 p. m. Subject to be announced but to be related to diseases of the kidney. Dr. Albert A. Hornor.

MAY — Annual meeting.

The censors meet on the first Thursdays of May and November in each year.

NORFOLK SOUTH

Meetings held at 12 noon.

MARCH 3 — Norfolk County Hospital, South Braintree.

APRIL 7 — At the Quincy City Hospital.

MAY 5 — Annual meeting.

PLYMOUTH

Meetings will be held at 11 a. m. on March 17, April 21, May 19 and July 21.

SUFFOLK

MARCH 15 — Joint meeting with Boston Obstetrical Society.

WORCESTER

At the following meetings, except the annual meeting, dinner will be at 6:15, to be followed by business session and scientific program.

MARCH 9 — Memorial Hospital, Worcester.

APRIL 13 — Hahnemann Hospital, Worcester.

MAY 11 — Afternoon and evening, annual meeting. Place and schedule of program to be announced.

BOOK REVIEW

Artificial Fever: Produced by physical means; its development and application. Clarence A. Neymann. 294 pp. Springfield and Baltimore: Charles C Thomas, 1937. $6.00.

The advance in artificial fever as a means of treatment in many diseases has been so rapid that most of the literature has necessarily appeared in medical journals. Some of this, having been collected and evaluated, is now published in monograph form. The author, Dr. Neymann, was one of the pioneer workers in this field, having begun his investigations as early as 1927 and published his first paper September 1, 1929. It would appear that he was the first to use high-frequency currents in man for the purpose of treating disease. His work was done originally at the Cook County Psychopathic Hospital and later at the Northwestern University Medical School. His book consists of an adequate historical introduction to the subject, excellent descriptions of the technic of electropyrexia and well-written chapters on the physiology of hyperpyrexia due to various agents, the effects of artificial fever upon diseases such as dementia paralytica, other forms of syphilis, multiple sclerosis, chorea minor, arthritis, gonorrhea and asthma, and finally, an evaluation of his own results as compared with those of others. In addition, he gives an extensive bibliography up to January 1, 1937, listing over 550 references.

The book may be read with many points of view in mind. Those interested in the history of artificial fever will find an adequate account of not only the more recents methods based upon electricity, but the older ones, particularly balneotherapy, used from very ancient times. Of most interest, perhaps, in the historical field are his account of patients' bathing in a bath of hot water in Japan, where the body temperature is raised by this method to as high as 104°F., and the forgotten demonstration by Philipps, in 1883, that by immersing himself in hot water, he raised his own temperature to 103°F. Dr. Neymann gives Philipps the credit, therefore, of being the originator of hyperpyrexia produced by physical agents.

The physiologist will be interested in the excellent chapter on the bodily effects of hyperpyrexia, particularly in relation to the velocity of blood flow and the effects on the heart, sodium chloride excretion, blood volume, blood sugar, and so forth. All these subjects, and many others, are treated in considerable detail. Another section of the book, which concerns the technic of electropyrexia, deals, in charts and photographs as well as in the text, with the various types of methods which have been used in the last ten years. The author has tried out practically all the methods which are described, beginning with diathermy, first reported in 1929, and followed by radiothermy, electromagnetic induction, electric blankets, radiant heat and hot-air cabinets, with his final adoption, in the last two years, of a combination of air-conditioned cabinets and electromagnetic induction. This he believes to be the safest and surest way to raise the temperature of the body and keep it at a constant level as long as desired.

Finally, the book will be useful to the clinician, for there are individual chapters on separate diseases, giving the results of treatment. Dementia paralytica has the best record, for in early cases selected for treatment the recovery rate is very high, perhaps up to 80 per cent. With institutionalized and deteriorated patients, naturally, the rate is much lower, but even in this group recovery may reach as high as 30 per cent, and another 30 or 40 per cent will show decided improvement. After electropyrexia, the use of tryparsamide and other heavy-metal salts is recommended. The results of treating other forms of syphilis of the central nervous system are not quite so satisfactory, although the author believes that, in cases of tabes dorsalis, artificial fever is a safe and practical method of treatment that promptly alleviates the lancinating pains and crises in a large percentage of the cases. The treatment of primary optic atrophy due to syphilis is still, in the author's opinion, in an experimental stage, and no striking results have been obtained. The same may be said of the treatment of primary and secondary syphilis; although improvement is readily noted, it would seem as though artificial fever alone will not eradicate the disease. The author is somewhat encouraged by his treatment of cases of multiple sclerosis and feels that the results with hyperthermia are better than those with any other form of therapy. In chorea minor the disease is promptly aborted in the vast majority of the cases, although recurrences may occur in a few after the treatment is over. Dr. Neymann feels that artificial fever is at present the treatment of choice for Sydenham's chorea. It also seems to be a specific for the treatment of gonorrhea, but there are other factors to be considered and the elaborate technic of treatment tends to forestall the use of this method when easier forms of treatment are at hand. It is thought, in addition, that electropyrexia may have some influence on asthma.

In general, it is too early to evaluate this form of treatment in any of the diseases except, possibly, dementia paralytica. The author takes a broad stand on this point, in spite of his intensive work on this form of treatment and his great interest in it. Although the subject is advancing so rapidly that this monograph will soon be out of date and require a second edition, nevertheless the book is one of great value for its historical perspective and for its view of the subject at the present time, seen through the eyes of an expert. For the details of treatment, the physiologic effects, and a review of the literature, this book can be highly recommended. The author would be the first to admit that it is not the final word on the subject, for advances have already been recorded since he was able to finish his manuscript, a few months ago.